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THE OCCURRENCE OF COR PULMONALE IN CASES OF BRONCHIAL ASTHMA*

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THERE is no uniformity of opinion regarding the role of bronchial asthma in the causation of heart disease. Most authors hold that cardiac complications of this origin are uncommon.[1-3] On the other hand, it has been pointed out that bronchial asthma and its sequelae, pulmonary emphysema, often lead to right-ventricular hypertrophy and finally heart failure.[4-6] The number of autopsied cases with bronchial asthma reported in the literature, however, is not large, and not all writers have recorded full data on the heart. For this reason, it is of interest to report the cardiac findings in 15 cases of bronchial asthma that came to autopsy. In addition, 54 patients were studied clinically to determine the incidence of cardiac abnormalities of this origin.

ANALYSIS OF DATA

Forty-six of these 69 ambulatory and autopsied patients were men; 23 were women. Fifty-nine (85 per cent) were past forty years of age. Forty-two (61 per cent) had had asthma for ten years or longer. The average duration of the disease was thirteen years. The cases were selected on the basis of the history, physical examination, blood studies and skin tests. A family history of allergy was present in 26 cases (38 per cent). Skin tests were positive in 46 cases (67 per cent), and an eosinophilia of 5 per cent or more was found in 22 cases (32 per cent).

Clinical Data

The histories and physical examinations of the 54 nonautopsied patients were studied to determine the presence or absence of congestive failure. Although in most patients dyspnea was an outstanding symptom, it could not be established that cardiac weakness was a factor except in 1 patient, who later gave evidence of a cor pulmonale. Likewise, physical examination revealed no evidence of heart failure.

Teleoroentgenograms were made in 46 cases. In 34 (74 per cent) the heart shadow appeared normal, in 7 (15 per cent) it was enlarged, and in 5 (11 per cent) the left ventricle was blunted and prominent. Six of the 12 patients with some cardiac enlargement had hypertension; an obvious cause for the enlargement was not found in the remaining 6.

The velocity of blood flow was determined by the Decholin method‖ in 40 cases.[7] The average normal circulation time with this method is 15 seconds. The readings in 37 cases (92 per cent) were within normal limits (average 12.8 seconds); in only 3 cases was the circulation time prolonged (22.0, 22.2 and 26.0 seconds, respectively). These 3 patients showed no clinical evidence of congestive failure. The electrocardiograms in 1, however, showed abnormally inverted T waves, and in another there was prolongation of the QRS interval. It has been shown that there is a tendency for the velocity of blood flow to be slightly increased in emphysema,[8] and this was true in the vast majority of our cases. The occurrence of a prolonged circulation time in these cases, in the absence of any other cause, was considered suggestive evidence of beginning circulatory failure.

Electrocardiograms were available in 46 cases. Fifteen (33 per cent) of these were within normal limits. Thirty-one patients (67 per cent) showed the following abnormalities: prolongation of the QRS interval, 3 cases; inverted T waves in Leads 1 and 2, 7 cases; abnormalities in Lead 4, 3 cases; an abnormally deep Q wave in Lead 3, 2 cases; a deep S wave in Lead 2, 7 cases[9]; and auricular fibrillation, 1 case. Thirteen patients (28 per cent) showed left-axis and 6 (13 per cent) right-axis deviation. A tendency to right-axis deviation was

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‖Decholin-Sodium was kindly supplied by Riedel-de Haen, Incorporated, New York City.

further noted in 4 other patients (9 per cent). The only abnormal finding in the electrocardiogram in 6 cases was left-axis deviation, and in 2 cases right-axis deviation.*

Autopsy Data

Autopsy data were available in 15 patients who died during the course of this study. A summary of the clinical and postmortem findings in these cases is shown in Table 1. Three patients had had bronchial asthma for a period of three years or less, the remaining 12 for six years or longer. Five of the latter died in congestive failure (Cases 1–5), and 4 of these showed predominant hypertrophy of the right ventricle. These 5 patients with congestive failure showed no evidence of valvular disease. The coronary arteries revealed no points of narrowing or occlusion, and the myocardium no areas of fibrosis or infarction.

Six patients (Cases 6–11) died of intercurrent diseases. In 3 of these (Cases 6–8) the heart was normal; 1 patient (Case 9) had hypertensive and arteriosclerotic heart disease, and 2 (Cases 10 and 11) showed an unexplained increase in heart weight. Asthmatic paroxysms played a prominent part in the terminal state in 4 patients (Cases 12–15), and 1 of these showed a slightly dilated right ventricle.

Table 1. *Clinical and Autopsy Findings in 15 Cases of Bronchial Asthma.*

Case No.	Age	Sex	Duration of Asthma	Duration of Cardiac Symptoms	Duration of Edema	Heart Findings					Lung Findings				Cause of Death
						Heart weight	Ventricular thickness		Coronary arteriosclerosis*	Infarcts	Chronic bronchitis	Diffuse fibrosis	Abscesses	Microscopical emphysema	
							Right	Left							
			yr.	*yr.*	*mo.*	*gm.*	*mm.*	*mm.*							
1	53	M	12	1½	10	620	12	15–20	+	0	±	+	0	0	Cardiac failure; cor pulmonale.
2	58	M	16	3	½	520	12	22	+	0	+	0	0	±	Cardiac failure; cor pulmonale.
3	60	M	12	?	2	420	7–10	12	++	0	+	+	0	+	Cardiac failure; cor pulmonale.
4	51	M	6	?1½	1½	400	10	15	+	0	±	+	+	0	Cardiac failure; cor pulmonale.
5	50	F	18			340	3	8	+	0	±	+	0	0	Cardiac failure
6	75	M	6			200	5	14	++	0	0	0	0	0	General peritonitis
7	72	M	20			250	6	18	++	0	0	+	0	0	Bronchopneumonia
8	62	M	42			340	7	15	+	0	0	0	0	0	Cancer of colon
9	72	M	Many			520	10	25	++++	+	+	0	0	+	Cerebral hemorrhage
10	46	M	20			390	8	17	+	0	+	0	0	0	Penetrating ulcer of colon
11	60	F	8			420	4	12	++	0	0	0	0	0	Hypernephroma
12	60	M	28			360	4	14	++	0	++	0	0	0	Bronchopneumonia; asthma.
13	46	M	3			250	6	16	++	0	+	+++	0	0	Pneumothorax, asthma.
14	60	M	2			340	4	22	+	0	+++	+++	0	0	Asthma
15	52	M	¾			300	7	16	+	0	+	0	+	0	Bronchiectatic abscesses; asthma.

*+ = smooth intima of major coronary vessels; ++ = numerous atheromatous plaques but no narrowing; +++ = definite narrowing of the major vessels; and ++++ = one or more major vessels occluded.

Case Reports

Case 1. A 53-year-old man was admitted to the hospital on July 11, 1933, complaining of a productive cough, shortness of breath, nocturnal dyspnea and edema. Twelve years previously he began to have asthmatic attacks, which were more marked in warm and damp weather. These became progressively more frequent and severe. Two years before admission he began to complain of dyspnea on exertion and attacks awakening him at night. Ten months before admission he began to have swelling of the lower extremities and palpitation on slight exertion. All the symptoms became progressively more marked.

Physical examination on admission showed cyanosis of the lips and cheeks and a uniformly enlarged thyroid gland. The chest was barrel shaped and hyperresonant, and expiration was prolonged. There were numerous rhonchi throughout both lung fields and occasional rales at the bases. The heart sounds were slow and regular, and there was a systolic murmur at the apex. The liver edge was palpable 4 cm. below the costal margin. The blood pressure was 100/55. Roentgen examination showed a transverse cardiac diameter of 18.3 cm. and a chest diameter of 32.2 cm. The laboratory findings were normal except for a white-cell count of 14,200. The basal metabolic rate was +54 per cent on admission, but a subsequent determination was +16 per cent. It was assumed that the patient was suffering from congestive heart failure, probably due to coronary heart disease. A diagnosis of cor pulmonale secondary to bronchial asthma and emphysema was not made.

Total ablation of the thyroid gland was considered with the hope of relieving the cardiorespiratory symptoms. At operation the thyroid gland was found to be considerably enlarged. Early in the postoperative course the temperature rose and the respiratory distress became more marked. In spite of all therapeutic measures, the patient died on August 3.

The surgically removed thyroid gland, although enlarged, showed normal tissue on microscopic examination. There was no evidence of hyperplasia.

Autopsy (No. 33–70). Throughout both lung fields there were many small bronchiectatic abscesses and areas of emphysema. In the upper portion of the left lung there was a particularly large area of emphysema that com-

*Axis deviation was determined by the angle method: angle below 0° = left axis; angle above 90° = right axis; angle 80 to 90° = tendency to right axis.

municated with areas deeper in the lung tissue. Microscopic examination showed extensive fibrous thickening and round-cell infiltration of the alveolar septums. The heart weighed 620 gm. The right auricle was tremendously dilated. The wall of the right ventricle measured 1.2 cm. and that of the left 1.5 to 2.0 cm. in thickness. The coronary arteries showed no arteriosclerosis and were patent throughout. There was no fibrosis or evidence of infarction.

CASE 2. A 58-year-old man was admitted to the hospital on October 21, 1935, complaining of cough, shortness of breath and swelling of the legs. Cough had been present for many years. Three years before admission he began to have paroxysmal attacks of shortness of breath with wheezing, which became progressively more frequent and severe. Two months before admission he noticed palpitation and orthopnea, and 6 weeks later edema of the legs appeared for the first time.

Physical examination on admission showed cyanosis, dyspnea and orthopnea. The chest was emphysematous, and there were dullness and coarse rales at both bases. The heart sounds were distant and rapid but regular. The second pulmonary sound was accentuated. There was pitting edema of the lower legs and over the sacrum. The blood pressure was 120/110. The laboratory findings were within normal limits. A diagnosis of congestive failure probably due to arteriosclerotic heart disease was made.

The patient was immediately placed in an oxygen tent, digitalized, and given sedatives. He did not respond to treatment, however, and died the next morning.

Autopsy (No. 35–92). The left lung weighed 500 gm., the right lung 640 gm. Gross sections through the right lower and middle and left lower lobes showed small saccular areas, 2 to 6 cm. in diameter, full of mucopurulent material. Microscopic examination showed dilatation of the small bronchioles and desquamation of the lining epithelium. Surrounding these small bronchioles there was much fibrosis and round-cell infiltration. The heart weighed 520 gm. The myocardium showed no evidence of softening, fibrosis or infarction. The coronary vessels were patent throughout, and their internal surfaces showed only a few small scattered atheromatous plaques. The wall of the right ventricle measured 1.2 cm. and that of the left 2.2 cm. in thickness.

CASE 3. A 60-year-old man was admitted to the hospital on August 24, 1937, because of marked weakness, precordial pain on exertion, increasing dyspnea, and peripheral edema of 9 weeks' duration. He had had attacks of bronchial asthma for 12 years, and for many years before this a nonproductive cough. Seven years before admission he had an attack of hemoptysis, for which he was admitted to a sanatorium. Six months later he was seen in the outpatient Allergy Clinic for cough and wheezing. Physical examination at that time showed cyanosis, prolonged and labored expiration, rhonchi throughout both lung fields and crackling rales at both apices. The heart action was slow and regular, and the sounds normal. There were no murmurs. The vital capacity was 1800 cc. Skin tests were positive for house dust. During the next 6½ years the patient was followed in the Allergy Clinic, but in spite of treatment, cough and wheezing continued. Approximately 2 months before admission, dyspnea and weakness became more marked and for the first time swelling of the lower extremities appeared.

Physical examination on admission showed orthopnea, dyspnea and cyanosis. Both lung fields were filled with musical and moist rales. In the right chest, below the scapula, there were amphoric breathing and cavernous voice sounds. The heart sounds were rapid but regular; there were no murmurs. The blood pressure was 106/68. The liver edge was felt two fingerbreadths below the right costal margin. There was marked pitting edema of both lower extremities to a point below the knees. The white-cell count was 23,000, and the blood nonprotein nitrogen 53 mg. per 100 cc. An electrocardiogram showed right-axis deviation.

In spite of digitalization and an oxygen tent, dyspnea, orthopnea and cyanosis became progressively more marked, and death occurred on the 3rd hospital day, following complete circulatory collapse.

Autopsy (No. 37–83). The right lung weighed 710 gm., and the left lung 560 gm. The apical surfaces were covered with thick plaques of fibrous tissue. The bronchioles were dilated and ended in saccular dilatations filled with purulent material. Microscopic sections showed a picture of old fibrosis, newly proliferating fibrous tissue and bronchiectatic cavities. The heart weighed 420 gm. The right ventricle appeared to be hypertrophied. The coronary arteries showed a few arteriosclerotic plaques, which did not greatly narrow the lumen. There were no areas of marked fibrosis or infarction. The wall of the right ventricle measured 0.7 to 1.0 cm. and that of the left 1.2 cm. in thickness.

CASE 4. A 51-year-old man was admitted to the hospital on February 20, 1931, because of progressive edema of the lower extremities of 6 weeks' duration. Approximately 8 years previously he began to have a chronic cough, which was later associated with wheezing and attacks of asthma. This condition grew gradually worse and was more marked in the fall and winter months. His father, who died at the age of 60, had had bronchial asthma. One and a half years before admission the patient was seen in the outpatient Allergy Clinic. At that time physical examination showed cyanosis, expiratory difficulty, a barrel-shaped, emphysematous chest and sibilant rhonchi throughout both lung fields. The vital capacity was 1300 cc. The heart sounds were slow and regular. The blood pressure was 120/90. Skin tests were positive for several common inhalants. During the next 18 months the patient was seen in the Allergy Clinic at weekly intervals. In spite of treatment he grew steadily worse, and was therefore admitted to the hospital.

Physical examination on admission showed dyspnea and marked cyanosis; the fingertips showed clubbing, the chest was emphysematous, expiration was difficult and markedly prolonged, and there were rhonchi throughout both lung fields. The heart sounds were slow, regular and of good quality. There were no significant murmurs. The blood pressure was 125/85. The liver edge was felt three fingerbreadths below the costal margin. The legs, thighs and scrotum were markedly edematous. Stereoroentgenographic examination showed no significant change in the lung fields. The transverse diameter of the heart, however, was markedly increased. An electrocardiogram showed marked right-axis deviation (angle 130°). All the laboratory findings were normal except for a white-cell count of 13,200. A diagnosis of congestive failure was made, but the presence of cor pulmonale due to bronchial asthma and emphysema was not suspected.

In spite of treatment, the patient showed no signs of improvement, and on the 5th hospital day he was found

in a state of collapse. Examination of the chest showed signs of consolidation in both lung bases posteriorly. He was placed in an oxygen tent. The temperature rose progressively to 104.0°F., tracheal rales developed, and the patient died on the 9th day after admission.

Autopsy (No. 31–20). There was a small amount of fluid in both pleural cavities. The lungs showed bilateral bronchiectasis with multiple bronchiectatic abscesses. The right lung weighed 1200 gm. and the left lung 590 gm. The right side of the right lower lobe was very firm, and on section this area showed a necrotic hemorrhagic abscess, 4 cm. in diameter and lined by a definite wall. The middle and upper lobes showed smaller but similar purulent areas. The left lung was somewhat collapsed. Throughout the various cut surfaces, especially in the lower lobe, grayish-white, firm raised areas were to be seen. The main bronchioles were distended and increased, and their thickened walls were traced to the periphery. Microscopic examination showed marked fibrosis, and the alveoli were lined by thickened fibrous walls. The bronchi were dilated and filled with pus. The heart weighed 400 gm. The musculature of the right side appeared almost as thick as that of the left. The wall of the right ventricle measured 1.0 cm. and that of the left 1.5 cm. in thickness. The interventricular septum showed no bulge to the right side. The coronary arteries showed no arteriosclerosis. There was no fibrosis or evidence of infarction.

Discussion

The effects of bronchial asthma and pulmonary emphysema on the right side of the heart are difficult to estimate clinically. Before congestive failure has occurred, hypertrophy of the right ventricle is usually not sufficiently marked to make itself evident by x-ray study. On the other hand, when congestive failure is present, the burden of proof is on those who maintain that the bronchial asthma or emphysema is of etiologic importance. Valvular, hypertensive and coronary disease must first be excluded, and to do this autopsy data must be available. The usual method of estimating ventricular hypertrophy by the thickness of the ventricular walls is at best crude, and it is probable that minor degrees of right-ventricular hypertrophy are not infrequently overlooked. This is particularly apt to be the case when heart disease is not the cause of death and the total heart weight is near the upper limits of normal. On the other hand, when at autopsy it is noted that the right ventricle is grossly hypertrophied and the thickness of its wall is well above the usual limits of normal, it is probable that right-ventricular strain of considerable magnitude had been present.

It is significant that many of the autopsied cases of bronchial asthma reported in the literature reveal unmistakable evidence of predominant hypertrophy of the right ventricle. Thus, of 5 autopsied cases with cardiac data reported by Huber and Koessler,[10] 2 had hypertrophy of the right ventricle. Harkavy[11] reported autopsy findings in 2 cases. One of these with bronchial asthma of only six months' duration did not show any cardiac abnormality. The second patient, however, a woman of thirty-four, who had had bronchial asthma for nine years, died of cor pulmonale. Five years before death she began to show progressive edema which reached enormous proportions. At autopsy the heart weighed 525 gm. The right ventricle and auricle were tremendously dilated and somewhat hypertrophied. The left ventricle was normal in size. Rackemann[12] reported a case of fatal asthma in a woman of forty-three with a history of asthma of twelve years' duration. At autopsy the heart weighed 290 gm., but the right ventricle was acutely dilated. MacDonald[13] reported cardiac findings in 8 autopsied cases. Three showed definite evidence of hypertrophy and in the remaining cases the right ventricle was dilated. Colton and Ziskin[4] reported autopsy data in 6 cases, and of these 1 showed marked hypertrophy and dilatation of the right ventricle. Sprague[6] reported 2 cases of fatal asthma with right-sided hypertrophy. Thieme and Sheldon[8] recorded cardiac weights and measurements of the thickness of the right and left ventricles in 7 of their autopsied cases. The asthma was of short duration (one or two years) in 3 of these patients. One of the remaining 4 with asthma of long duration showed definite hypertrophy of the right ventricle.

Recently, Kountz, Alexander and Prinzmetal[5] determined the weight of each ventricle separately in 17 cases of pulmonary emphysema. Since the end result of severe bronchial asthma is pulmonary emphysema, their data are germane to this problem. The cardiac findings were within normal limits in 7 cases. The remaining 10 showed predominant hypertrophy of the right ventricle. These authors reproduced similar cardiac findings in 19 dogs after the production of pulmonary emphysema experimentally. Eleven animals showed dilatation and predominant hypertrophy of the right ventricle.

The duration of bronchial asthma in 3 of our 15 autopsied cases was three years or less; the hearts were normal. In the 12 other cases, in which asthma had existed for six years or longer, 4 patients (33 per cent) showed unmistakable effects of right-ventricular strain and died in congestive heart failure. Heart failure may have been related to asthma in 1 other patient (Case 5), who showed no obvious hypertrophy of the right ventricle. Injection dissection studies by the Schlesinger technic[14] in this case revealed no evidence of coronary disease. The electrocardiogram showed right-axis deviation.

Although our group of 54 cases studied only clinically showed with one exception no evidence of heart failure, the electrocardiographic examina-

tion revealed many abnormalities that can probably be ascribed to myocardial disease. This finding is in accord with the studies of Unger,[15] Kahn,[16] Anthony[17] and Colton and Ziskin.[4] The most significant finding was right-axis deviation in 13 per cent of the cases (angle above 90°). In another 9 per cent, a tendency to right-axis deviation was present (angle 80 to 90°). This is particularly significant, for it has been shown that right-axis deviation producing an angle of 90° or over is regularly associated with predominant hypertrophy of the right ventricle. In a series of 68 normal hearts, for example, Proger and Davis[18] did not find a single case with an electrical axis above 80°. Of the patients in whom asthma was present for ten years or longer, the incidence of right-axis deviation was 18 per cent. Apparently, the longer the duration of asthma, other factors being equal, the greater the strain on the right ventricle and the higher the incidence of right-axis deviation. Right-axis deviation of the magnitude noted cannot be accounted for by alteration in the position of the heart due to a low diaphragm. These changes in the electrocardiogram are in harmony with the occurrence of right-ventricular hypertrophy at autopsy.

It would appear, then, that cardiac complications in the course of bronchial asthma and its sequelae, emphysema and bronchiectasis, are much commoner than is generally believed. When circulatory failure first appears, its diagnosis is often made difficult by dyspnea of emphysematous origin. Although some patients with cor pulmonale show edema for many months before death, others do not. In Case 2, for example, there was progressive dyspnea for three years. Peripheral edema and pulmonary congestion, however, were present for only a few weeks before death. Also in Cases 3 and 4 the presence of edema before death was of short duration. Thus, rapidly progressing heart failure may appear suddenly in patients with cor pulmonale.

Summary and Conclusions

The heart was studied in 69 patients with bronchial asthma; the autopsy findings in 15 of these cases are analyzed.

Electrocardiographic abnormalities, indicating right-ventricular strain and myocardial damage, were noted in a comparatively high percentage of patients with chronic bronchial asthma.

Five of 12 autopsied cases with bronchial asthma of six or more years' duration died in congestive failure, and 4 of these showed predominant hypertrophy of the right ventricle.

Cor pulmonale due to chronic bronchial asthma is a commoner disorder than is generally recognized.

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A DIETARY STUDY OF SUBJECTS FROM UPPER INCOME GROUPS*

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THIS dietary study was undertaken in 1940 because so little has been done on the dietary pattern of private patients. We decided, therefore, to analyze the diets of private patients to determine their daily intake of dietary essentials. We also considered it worth while and interesting to record the number of adults, from the same income level, but not under active medical supervision, who daily consumed the foods that the Committee on Foods and Nutrition of the National Research Council has stated every person must have to secure the required amounts of dietary essentials.

MATERIAL AND METHODS

Two hundred and twenty-five patients, 92 males (9 of whom were physicians) and 133 females, of various ages, from the upper income brackets and representing a cross-section of private patients attending our clinic, were selected for this study. In addition, 223 persons (20 of whom were physicians), from various upper-middle-class groups that we addressed from time to time, answered questionnaires regarding their dietary pattern; they were not, so far as we knew, under medical supervision.

For each of the 225 patients, we had a complete dietary history, including food habits, dislikes, intolerances and cravings, and a seven-day dietary calculation formulated on the basis of a diet questionnaire filled out by each patient — including the amounts and kinds of all food and liquid ingested, both during and between meals, and the mode of preparation of food. The patients were given these questionnaires when they first presented themselves with their complaints, and were told to fill them out meal by meal for a week, expressing quantities in cupfuls, tablespoonfuls, teaspoonfuls, ounces or number of units, such as one tomato, half a tomato and so forth. When they returned a week later with their questionnaires filled out, we went over them with the patients, making certain that salad dressings, sugar and cream, and the like were recorded. While reviewing their diets, we also suggested dietary changes in line with their specific ailments.

Since exact weights of the amounts of food consumed were not recorded, we realize that our findings may not have been completely accurate. However, we are of the opinion that, had we asked the patients to weigh their food, we should have had little co-operation. Furthermore, although the amounts recorded were not completely accurate, we believe that we have a true picture of the patients' dietary patterns and that in these patterns dietary deficiencies were evident. Whether a diet contained 3800 or 4000 international units of vitamin A made little difference, the important fact being that the diet was deficient in vitamin A.

The diets were calculated to determine the amounts of protein, fat, carbohydrate, calories, vitamins A, B_1, B_6 and D, ascorbic acid, riboflavin, niacin, calcium, phosphorus, iron, copper, potassium, sulfur, magnesium, manganese, sodium, chlorine, acid-base, fiber and water. The food values were obtained chiefly from Bowes and Church's *Food Values of Portions Commonly Served*, both the third and the fourth edition.[1] While we were still in the process of calculating diets, Bowes and Church published the fourth edition of their book and we proceeded to make our calculations from this edition. In addition to Bowes and Church's text, we used Hawley and Maurer-Mast's.[2] We modified many of Bowes and Church's figures in their third edition in accordance with Munsell's[3] work, and went to the original literature[4, 5] for calculations on the trace minerals. Unfortunately, our data for the trace minerals and for niacin and vitamin B_6 were not complete because of lack of information on their content in many foods. While we were using Bowes and Church's third edition, we expressed vitamin B_1 and riboflavin in terms of international units and Sherman-Bourquin units respectively; when we went to their fourth edition, we used their revised method of expression — vitamin B_1 and riboflavin in micrograms.

After the patients' diets were calculated for their weekly intakes, we divided by 7, to determine the average daily intakes. We did not calculate daily intake of trace minerals, vitamin B_6 and niacin, since our data were incomplete; we merely computed the weekly intake of these factors. The diets were then scored for each of the dietary essentials — protein, calories, vitamins A and B_1, riboflavin, ascorbic acid, calcium, phosphorus and iron — on the basis of the standards set up by the Committee on Foods and Nutrition of the National

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Research Council,[6] taking into consideration age, sex and activity (Table 1). These are tentative allowances and can be met by a good diet of natural foods, which will also provide other minerals and vitamins whose requirements are less well known.

A deviation of 5 per cent was allowed for all the essentials except calories, for which we allowed 10 per cent. We established the following standards for the other constituents of the diet in line with scientific research: carbohydrate, 50 to 60 per cent of the total calories[7]; fat, 20 to 35 per cent of the total calories[8]; acid-base, both the same or slightly more acid than base[9]; fiber, 5.0 to 6.3 gm.[10]; and water, 2800 cc.[11] For carbohydrate and fat, we allowed a 5 per cent, and for fiber and water, a 10 per cent deviation. Since our data were incomplete for the trace minerals, vitamin B_6 and niacin, we did not score the diets on these components. Regarding sodium and chlorine, we questioned the patient concerning the amount of salt used. If the amount was stated as "average," we estimated the salt intake, in addition to the amount found in food, as 6 gm. of sodium and 6 gm. of chlorine daily; if the amount was "large," we estimated the daily sodium and chlorine as 8 gm. respectively and if the amount recorded was "little," as 4 gm. respectively. Since we could not calculate the amount of vitamin D derived from sunshine, we merely recorded the amount of vitamin D in the weekly diet.

We then accumulated our data on these 225 diets to determine how many were deficient in vitamin A, how many were deficient in vitamin B_1 and so forth, and to determine their degree of deficit. For the trace minerals, vitamin B_6, niacin and vitamin D, we expressed our findings in terms of numerical ranges.

We also recorded how many diets had only one deficiency, how many had two deficiencies and so forth. In accumulating these data, we considered only protein, calories, calcium, phosphorus, iron, vitamins A and B_1, ascorbic acid and riboflavin, since these are the dietary factors that are principally concerned. We have eliminated the other factors for the following reasons: the amount of fat and of carbohydrate, because little is known about the ill effects of too much fat (over 35 per cent of the total calories) or of too little carbohydrate; acid-base balance, because it is uncertain whether the balance of acid-forming and base-forming elements in food is of practical significance

TABLE 1. *Recommended Daily Allowances for Specific Nutrients.*[9]

	Energy	Protein	Calcium	Iron	Vitamin A*	Thiamin (Vitamin B_1)†	Riboflavin	Nicotinic Acid	Ascorbic Acid† (Vitamin C)	Vitamin D¶
	cal.	*gm.*	*gm.*	*mg.*	*int. units*	*mg.*	*mg.*	*mg.*	*mg.*	*int. units*
Man (70 kg.)										
Moderately active	3000	70	0.8	12	5000	1.8	2.7	18	75	
Very active	4500					2.3	3.3	23		
Sedentary	2500					1.5	2.2	15		
Woman (56 kg.)										
Moderately active	2500	60	0.8	12	5000	1.5	2.2	15	70	
Very active	3000					1.8	2.7	18		
Sedentary	2100					1.2	1.8	12		
Pregnant (latter half)	2500	85	1.5	15	6000	1.8	2.5	18	100	400–800
Lactating	3000	100	2.0	15	8000	2.3	3.0	23	150	400–800
Children up to 12 years										
Under 1 year‡	100/kg.	3–4/kg.	1.0	6	1500	0.4	0.6	4	30	400–800
1–3 years§	1200	40	1.0	7	2000	0.6	0.9	6	35	
4–6 years	1600	50	1.0	8	2500	0.8	1.2	8	50	
7–9 years	2000	60	1.0	10	3500	1.0	1.5	10	60	
10–12 years	2500	70	1.0	12	4500	1.2	1.8	12	75	
Children over 12 years										
Girls										
13–15 years	2800	80	1.3	15	5000	1.4	2.0	14	80	
16–20 years	2400	75	1.0	15	5000	1.2	1.8	12	80	
Boys										
13–15 years	3200	85	1.4	15	5000	1.6	2.4	16	90	
16–20 years	3800	100	1.4	15	6000	2.0	3.0	20	100	

*Requirements may be less if provided as vitamin A; greater if provided chiefly as the provitamin carotene.

†1 mg. thiamin = 333 int. units; 1 mg. ascorbic acid = 20 int. units.

‡Needs of infants increase from month to month; the amounts given are for approximately 6 to 8 months. The amounts of protein and calcium needed are less if derived from breast milk.

§Allowances are based on needs for the middle year in each group (2, 5, 8 and so forth) and for moderate activity.

¶Vitamin D is undoubtedly necessary for older children and adults; when not available from sunshine, it should be provided probably up to the minimum amounts recommended for infants.

in human nutrition[12]; fiber, because individual needs and methods of cookery vary greatly and influence the requirement and the content of fiber in foods; and other factors, because the data were incomplete.

After analyzing over two hundred diets, we could deduce merely from looking over the weekly diets whether or not the dietary essentials were provided in adequate amounts. For example, if a pint of milk was not ingested daily, the diet would be deficient in riboflavin, and probably also in calcium, since the amount of calcium present in other sources, such as vegetables, is not adequately utilized and is variable, depending on the calcium content of the soil; if white bread was taken instead of enriched white or whole-wheat bread, the diet would be deficient in vitamin B_1; if the amount of meat ingested was small, the diet would probably be deficient in riboflavin (unless a good deal of milk was consumed), phosphorus and iron; and if there were no citrus fruits or raw cabbage or green pepper or raw green leafy vegetables, the diet would be faulty in ascorbic acid.

We then analyzed the dietary "yardstick" adopted during the National Nutrition Conference,[13] and found that a daily consumption of at least one pint of milk, three slices of whole wheat (or enriched) bread or its equivalent in whole-grain cereal, one serving of meat, one egg, two servings of vegetables, two servings of fruits (one of which should be a citrus fruit) and two and a half tablespoonfuls of butter, cream or fortified oleomargarine,

TABLE 2. *Number of Cases of Dietary Imbalance in 225 Private Patients.**

TYPE OF IMBALANCE†	NO. OF CASES AND DEGREE OF IMBALANCE						TOTAL NO. OF CASES
	5–10%	10–20%	20–30%	30–40%	40–50%	+50%	
Protein deficiency	21	23	17	16	3	2	82 (36.5%)
Fat deficiency	–	–	–	–	–	–	0 (0.0%)
Fat excess	29	39	65	43	16	8	200 (88.0%)
Carbohydrate deficiency	48	72	56	15	7	2	200 (88.0%)
Carbohydrate excess	1	–	–	–	–	–	1 (0.4%)
Calorie deficiency	–	33	50	44	29	11	167 (74.0%)
Calorie excess	–	5	5	–	–	–	10 (4.4%)
Vitamin A deficiency	10	12	12	10	3	11	58 (26.0%)
Vitamin B_1 deficiency	8	22	41	37	35	29	172 (76.0%)
Riboflavin deficiency	10	19	22	30	35	58	174 (77.0%)
Ascorbic acid deficiency	1	8	6	3	1	10	29 (13.0%)
Calcium deficiency	11	20	13	23	8	29	104 (46.0%)
Phosphorus deficiency	9	12	20	11	1	1	54 (24.0%)
Iron deficiency	14	27	26	11	10	2	90 (40.0%)
Water deficiency	–	25	41	30	39	28	163 (72.0%)
Water excess	–	–	–	–	2	1	3 (1.3%)
Fiber deficiency	–	31	27	35	26	36	155 (69.0%)
Fiber excess	–	6	4	–	–	–	10 (4.4%)

*Patients eating between meals numbered 94, or 42 per cent; this figure does not include those who ate fruit, fruit juices or milk, which do not interfere with appetite.

†In addition, 161 patients, or 71.5 per cent, had an acid-base imbalance; of these, 155 had more base-forming and 6 more acid-forming foods.

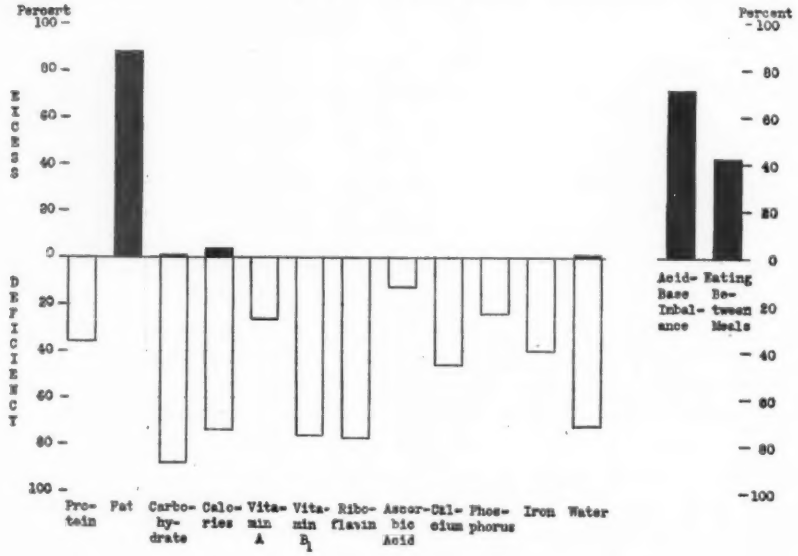

FIGURE 1. *Summary of Patients' Dietary Imbalances.*

provided the following essentials: 1500 calories, 62.2 gm. of protein, 0.85 gm. of calcium, 17.2 mg. of iron, 4300 international units of vitamin A, 1400 microgm. of Vitamin B_1, 80 mg. of ascorbic acid, and 1700 microgm. of riboflavin. A comparison with the standards reproduced in Table 1 indicates how many of the dietary essentials are furnished by this dietary yardstick, which supplies less food than the average person ingests.

We then decided that it would be worth while to compare the diets of patients and healthy persons, to obtain a picture of the dietary deficiencies of the upper income groups. We therefore distributed questionnaires to members of lay and professional groups whom we addressed from time to time, asking them to record "yes" or "no" to questions concerning their daily ingestion of the foods listed in the dietary yardstick. The answers to these queries were recorded and compared with the dietary pattern of the patients.

Dietary Findings in Private Patients

The dietary computation in 225 private patients is shown in Table 2.

Figure 1 illustrates graphically the percentage range of these dietary deficiencies and faulty habits. The values for other factors are expressed in numerical ranges on a weekly basis (Table 3). Although little is known of the functions in the body of the trace minerals and vitamin B_6, clinical evaluation of such factors will undoubtedly be accomplished in the future, and perhaps, these ranges will be of value to other investigators. However, it is known that manganese is necessary for the synthesis of vitamin B_1 and ascorbic acid, that a deficiency of copper may produce anemia, and that excess fluorine causes dental caries.

It should be noted that the diets of the majority of patients were high in fat (Fig. 1). We believe this excess is linked with large amounts of money to spend for food, since butter, cream, creamed vegetables, rich desserts and the like are relatively expensive. We have found that persons in the lower income levels cannot afford to spend large amounts of money on fat, and as a result their diets are significantly lower in fat.

Diets of Physicians

Among the 225 patients were 9 physicians whose diets were as bad as those of the other patients. Two of the 9 were deficient in protein; all had an excess of fat; all were deficient in carbohydrates; 7 were deficient in calories; 4 were deficient in vitamin A; all were deficient in vitamin B_1; 8 were deficient in riboflavin; 1 was deficient in ascorbic acid; 4 were deficient in calcium; 1 was deficient in phosphorus; 5 were deficient in iron; 4 had acid-base imbalance; 7 were deficient in water; and 3 ate between meals.

Preparation of Food

According to the patients' answers to questions regarding vegetable cooking, in most cases the vegetables were not prepared or cooked properly. A good deal of water was used in cooking, the remaining pot liquor was not used, potatoes were not cooked in their jackets and so forth. Therefore, in most cases, because of losses in cooking, the diets of the patients undoubtedly contained less vitamins and minerals than were found on calculation.

Table 3. *Other Factors in Diets of 225 Private Patients.*

Factor	Amount			No. of Weekly Diets
	int. units	*mg.*	*gm.*	
Vitamin B_6	0–1750			43
	1750–3500			62
	3500–5250			52
	5250–7000			36
	Over 7000			32
Niacin		0–35		174
		35–70		31
		Over 70		20
Vitamin D	0–175			40
	175–350			104
	350–525			60
	525–700			11
	Over 700			10
Copper		0–5		76
		5–10		118
		10–15		23
		15–20		6
		Over 20		2
Potassium			0–5	20
			5–10	78
			10–15	87
			15–20	27
			Over 20	13
Magnesium			0–1	115
			1–2	99
			2–3	11
Manganese			0–5	48
			5–10	91
			10–15	45
			15–20	25
			Over 20	16
Sodium*			Below 30	2
			30–40	29
			40–50	132
			50–60	46
			Over 60	16
Chlorine*			Below 30	1
			30–40	21
			40–50	71
			50–60	111
			Over 60	21

*Includes amount used in table salt.

Multiplicity of Dietary Deficiencies

It must be remembered that vitamin deficiencies are multiple and are rarely found alone, not only because a diet that is deficient in one factor is usually deficient in more than one factor, but also because a deficiency of one factor causes a greater strain on the other factors and, in turn, brings about an increase in the body's demand for these.

On compiling the data concerning the number of diets that were deficient in one or more of the essential factors,—protein, calories, vitamins A and B_1, ascorbic acid, riboflavin, calcium, phosphorus and iron,—we found the following: diets deficient in 0 factors, 28, or 12 per cent; diets deficient in 1 factor, 21, or 9 per cent (1 physician); diets deficient in 2 factors, 42, or 18 per cent; diets deficient in 3 factors, 41, or 18 per cent (4 physicians); diets deficient in 4 factors, 23, or 10 per cent (2 physicians); diets deficient in 5 factors, 21, or 9 per cent; diets deficient in 6 factors, 16, or 7 per cent; diets deficient in 7 factors, 18, or 8 per cent (2 physicians); and diets deficient in all factors, 15, or 7 per cent.

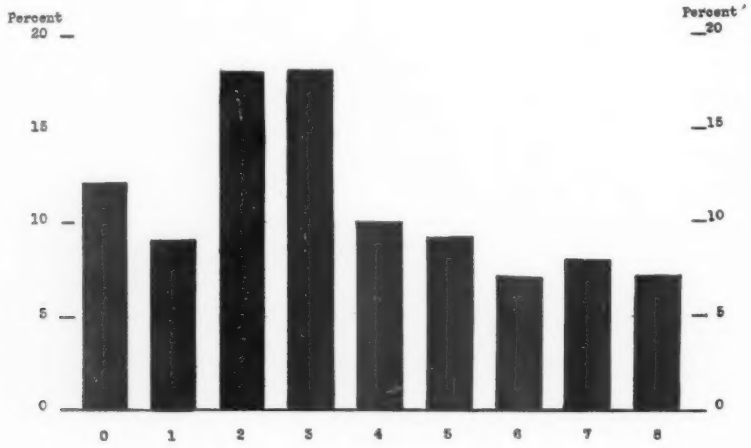

FIGURE 2. *Incidence of Number of Essentials in Which Patients' Diets Were Deficient.*

Of the 21 diets deficient in 1 factor, 9 were deficient in vitamin B_1, and 8 in riboflavin; of the 42 diets deficient in 2 factors, 36 were deficient in both B_1 and riboflavin (Fig. 2).

Dietary Findings in Presumably Healthy Persons

We endeavored to determine how closely the dietary pattern of our group of 225 private patients conformed with that of a group of 223 nonmedical subjects of similar socioeconomic status. To accomplish this, we recorded the answers of the "nonpatients" to a series of questions concerning their daily ingestion, in proper amounts, of the foods listed in the dietary yardstick.[13] The data thus obtained are presented in Figure 3.

In a survey of this sort, many errors are incurred. For example, we are convinced that not many people eat whole-wheat or enriched bread. This has been proved by the calculation of the diets of our patients and also by the fact that the consumption of whole-wheat bread is very small and that at the time this survey was made only a small fraction of the white bread sold on the market was enriched. Yet the figures concerning the general consumption of whole wheat or enriched bread belie this statement. We are certain that the nonmedical subjects of our survey often answered "yes" to many questions in the questionnaire because they knew the answer should be "yes." However, for purposes of comparison, we found these questionnaires valuable.

Discussion

In this study we observed that more of the diets of the private patients were deficient in vitamin B_1 and riboflavin than in any of the other vitamins or minerals. A recheck of the foods eaten or not eaten revealed that daily quotas of milk (one pint), whole-wheat or enriched grain products (two or three slices of bread or its equivalent in cereal), eggs (three to five a week) and fruit (two servings) were supplied by the diet *less often* than those of other nutritionally essential foods were.

Since the diets were often low in milk, they were of necessity low in riboflavin (and also calcium), and being low in whole-wheat or enriched grain products, they were low in vitamin B_1. The lack of eggs is not clearly illustrated by a definite deficiency in the diet, for the various minerals and vitamins contained in eggs can be supplied by other foods. In most cases, one serving of fruit was consumed daily. The lack of a second serving did not result in a specific dietary inadequacy, since the large consumption of vegetables made up for this lack. Because the consumption of fat, citrus fruits and vegetables was high, there were but few deficiencies in vitamin A and ascorbic acid. Since the protein intake in

many cases was low, iron and phosphorus deficiencies appeared.

A comparison of the patients' diets with the diets of nonpatients indicated that the dietary pattern of the latter was about the same as that of the former. In the questionnaires returned by the nonpatients, there were more "no" answers to queries concerning adequate consumption of milk, eggs, fruit and whole-wheat or enriched grain products than to those concerning the other essential foods. The water intake was low in both groups. If these diets could be calculated quantitatively, deficiencies of riboflavin and vitamin B_1 undoubtedly would rank highest. Most of the nonpatients obtained their one serving of meat daily, but only one serving of meat each day does not satisfy the protein requirement because the milk and, possibly, the eggs are needed to supply the balance.

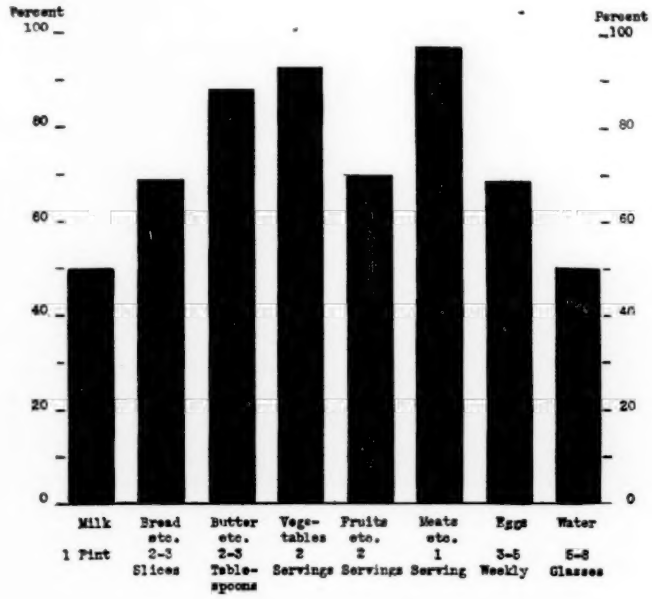

FIGURE 3. *Summary of Nonpatients' Daily Dietary Pattern.*

After questioning patients and nonpatients, we found that there were many reasons why people did not drink milk. Milk was considered fattening, milk made them constipated, milk gave them indigestion and so forth. We believed that some somatic condition caused an intolerance to milk and thought that this intolerance might be linked with a condition of achlorhydria. We therefore made a survey of 597 patients who attended this clinic, checking the patients' dislike for milk and the hydrochloric acid content of their gastric juices. Unfortunately, there seemed to be no correlation. Of 80 patients who had histamine achlorhydria, only 20 disliked milk; of 311 with hypochlorhydria, 70 disliked milk; and of 206 with euchlorhydria (normal hydrochloric acid content), 40 disliked milk. However, we believe that some still undetermined condition in the large number of people who dislike or do not tolerate milk is correlated with such intolerance.

SUMMARY AND CONCLUSIONS

The diets of 225 private patients from the upper income level, 9 of whom were physicians, were calculated.

Questionnaires regarding dietary pattern were filled out by 223 persons from the upper income level, 20 of whom were physicians, but none of whom were under direct medical supervision.

Among the private patients, the most prominent dietary deficiencies were in vitamin B_1 and riboflavin—76 per cent of the patients being deficient in vitamin B_1 and 77 per cent deficient in riboflavin.

Among those subjects not under direct medical supervision, milk, eggs, fruit and whole wheat or enriched grain products were the foods most frequently found lacking. This dietary pattern was similar to that of the patients, and on calculation, the diets of these 223 individuals would undoubtedly also be shown to be primarily deficient in vitamin B_1 and riboflavin.

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THE TREATMENT OF COMMON SKIN DISEASES*

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THE skin conditions that we see most commonly at the Vanderbilt Clinic and on the wards of the Presbyterian Hospital are acne, psoriasis, dry chapped skin, coccal infections, fungus infections and allergic dermatoses.

The first step in the treatment is the diagnosis. In addition to the appearance of lesions, there are three factors of distinct diagnostic help: the patient's story — of particular importance in the allergic group; laboratory tests, such as the direct examination of the involved skin for fungi or cocci and the use of skin tests in the diagnosis of the allergic group; and the therapeutic test, that is, specific treatment for the most likely diagnosis.

ACNE

A very mild acne is almost normal with the onset of puberty. Mild to moderately severe acne can be well treated by the general practitioner, but severe cystic and nodular acne requires the specialist's care. The treatment may be divided into local and systemic. Local care includes meticulous and prolonged work. At bedtime the areas involved should be thoroughly scrubbed with either tincture of green soap or sulfur soap. The comedones should be expressed with a blackhead remover and then sulfur — either Lalotin (Lascoff) or Sulpholac (Alba) — should be massaged into the areas in which the blackheads have been removed. In the morning the sulfur is removed by thorough scrubbing with warm water and soap. This is followed by rinsing with cold water. Powder may be used, but all creams and greases should be avoided. The systemic treatment of acne should include normal adequate food intake and the avoidance of chocolate, iodized salt and an excess of sweets and greasy foods; a normal amount of exercise and sunshine; and the correction of any systemic disorder, such as constipation or dysmenorrhea. The discrete use of endocrine preparations in patients having clear-cut menstrual difficulties is worthy of trial.

PSORIASIS

As in the treatment of acne, the therapy of psoriasis resolves itself into local and systemic. Dermatologists no longer believe that the patient with psoriasis need go about feeling that nothing can be done for him. Much can be done with purely local therapy, such as crude coal tar and the ultraviolet treatment of Goeckerman.[1] However, there is an increasing literature suggesting that psoriasis is a disturbance of lipid metabolism. Since 1933 a number of reports have appeared in the German literature on the successful treatment of psoriasis by rigid adherence to a diet not exceeding 20 gm. of fat daily. Later Dragstedt and his coworkers[2] reported that 17 out of 23 patients became free from psoriasis following the ingestion of lipocaic. This lipotropic substance, which is isolated from the pancreas, prevents or cures the fatty liver that develops in depancreatinized dogs maintained on insulin. Since 1939 we have been trying various lipotropic substances, such as crude liver, rice polish, yeast concentrate, soy-bean phosphatides and defatted wheat germ, in the treatment of psoriasis. With the use of the last two we have had astonishingly good results, particularly in the early type of lesions. The soy-bean phosphatide preparation that we have used is Lexo Cookies (American Lecithin Company); the patients eat from two to five of these daily. The defatted wheat germ is called Vio-Bin (Vio-Bin Corporation); one to two tablespoonfuls are eaten daily.

Along with the lipotropic substances the local medication has been one of the newer tar preparations — 3 per cent Nu-kol-tar (Benet) or 5 per cent Neo-tar (Lascoff) ointment. Many dermatologists use one of the di-oxyanthralin derivatives, such as 0.1 to 0.5 per cent Anthralin (Abbott) ointment.

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Two generalizations may be made concerning the local treatment of psoriasis. In an acute outbreak only soothing applications should be used, and when the eruption is chronic the lesions should be stimulated with one of the preparations mentioned above. Furthermore, at the beginning the various medicaments should be of average or below-average strength, and the strength increased if necessary; for example, one may begin with 0.1 per cent Anthralin and progress to 0.5 per cent. The older vehicles — lanolin, benzoated lard, petrolatum and so on — are being rapidly replaced by wetting agents in water-soluble bases. In these may be incorporated most local medicaments. They are nongreasy, much more penetrating, and wash off readily. At present there are three excellent ones; Solucreme (Lascoff), Hydrosorb base (Abbott) and Mull-sol (Hickok).

Dry, Chapped Skin

Dry, chapped skin is very prevalent in the winter time among people with rather thin skins, and those who wash their hands frequently, such as physicians. Sometimes in addition sharply defined patches of eczema develop on the extremities.

The skin should be treated gently. Soft water should be used if available. Mild or superfatted soaps, or the newer detergents, such as Lavol (Hickok) and Terses (Doak) are to be preferred. They are less hard on the skin than the ordinary soaps. The soap should be rinsed off and the skin carefully dried, and anointed with oil or cold cream at bedtime. Vitamin A is almost a specific for very dry skin. It is well to stress foods high in vitamin A and in addition to prescribe 75,000 units of vitamin A a day for a few weeks and then reduce it gradually to 25,000 units a day for a few months.

Coccal Infections

For staphylococcal and streptococcal infections of the skin — the commonest, perhaps, being impetigo — treatment with sulfathiazole is simple. Formerly a child with such an infection was excluded from school for weeks, but now it is possible for him to return to school within three or four days. One can use either 5 per cent sulfathiazole in a water-soluble base containing a wetting agent, or, if glycerin is available, about 8 parts of warm glycerin to 1 part of sulfathiazole. Either of these should be applied to the lesions every three or four hours.

We have not had good results with the local use of sulfonamides for boils. This is understandable because of the presence of pus.

For chronic, recurrent boils, dermatologists have a therapy different from that of surgeons or roentgenologists. In the few cases which reach the dermatology clinic we have found staphylococcal toxoid of remarkable benefit. Its use seems logical because the patient has insufficient resistance to an organism found on the skin. An effort is made to stimulate antitoxin production. Intradermal injections of staphylococcal toxoid (Lederle) are given three times a week. The average initial dose is 0.1 cc. of a dilution containing 1000 necrotizing units per cubic centimeter. The dose is increased by 0.1 cc. each time up to 0.7 cc. Then the next stronger dilution (10,000 units) is used and the course is repeated.

With this type of therapy, the boil may come to a head or may be reabsorbed. Usually no more boils occur after the patient has had a few injections.

Fungus Infections

It has been suggested that the best way to cope with superficial fungus infections is to let everyone have such an infection and learn to live with it. There are two types of causative organisms; dermatophytes, affecting the skin, hair and nails; and yeastlike fungi, the monilias, affecting the skin, nails and mucous membranes.

Perhaps the most effective therapeutic agent is gentian violet. If there is a tender, excoriated or fissured area, a 2 per cent aqueous solution can be used. If not, 2 per cent gentian violet in 25 per cent alcohol is preferable. Once the lesion becomes chronic, a medicament is necessary that will cause exfoliation. Whitfield's ointment is as good as any. For a fungicidal powder, B.F.I. (Mulford), is acceptable.

Reinfection in ringworm infection is probably not very common. There is, rather, a persistent infection, the site being usually the nails of the great toes. If they are serrated, discolored, too thick or too thin, the fungus may be there, and it is well to keep on treating the nails with alcoholic gentian violet.

For the differential diagnosis, it is quite easy to obtain the fungus or the yeastlike organism on culture or to recognize it by direct slide examination. When it is a question of whether the condition is a fungus infection, contact dermatitis or something else, one should take advantage of this laboratory procedure.

With ringworm infections the stockings should be sterilized. Of course, cotton is easily sterilized, but silk or wool is not. The best thing to do is to launder the hose and press them on the wrong side with a hot iron. Shoes do not need to be sterilized if stockings are worn. Bedroom slippers

can be discarded, or sterilized by placing small open jars of formalin in them for a few hours.

Allergic Dermatoses

Drug Eruptions

Drug eruptions are becoming an increasing problem. There are about two hundred drugs in common use. Unfortunately, any drug may produce an eruption, and practically every known dermatologic condition can be produced by them. A few of them are characteristic. The diagnosis of a drug eruption is made by its sudden appearance, its symmetrical distribution, its extensiveness in comparison with the systemic disturbance, and its brightness.

The history of drug ingestion is sometimes unobtainable; however, the patient has usually been taking the drug for some time. It seems desirable to dispense with some of the drugs now in use, at least some of the sedatives. A walk around the block or a hot bath may be advised instead. The treatment consists in prohibiting the use of the drug and in allaying the itch with sodium bicarbonate baths and calamine lotion.

There are few specific treatments for drug eruptions, with the exception of bromide and iodide eruptions, for which we give sodium chloride. In persons known to be sensitive to arsphenamine, it has been possible to give vitamin C prophylactically. Even after the eruption is present, the use of vitamin C is helpful.

Allergic Eczema

Allergic eczema is commonly seen in infants. It occurs on the flush areas, over the head and neck, and it may become widespread. It may clear up or persist. When it recurs in early childhood, it has no very characteristic distribution except that it is symmetrical and patchy. In adolescence and adult life, a characteristic picture is present: it is perfectly obvious that the skin has been scratched. The eczema occurs on the face and neck and in the antecubital and popliteal creases. For these adults with persistent eczema, dermatology clinics have little to offer. But we are most anxious to treat the infant or the child who has an eczema that has persisted more than six months in order to determine the substances to which he is sensitive.

Allergic eczema is primarily a gastrointestinal sensitivity and, unfortunately, the infant is usually sensitive to the foods he needs the most; egg white, cow's milk, wheat, orange, tomato, chocolate and so on.

The most helpful thing to the physician is the child's history. Often an intelligent mother will say that shortly after ingestion of a food, the child refused it. She insisted on it, and the rash developed. Or a mother will say, "Almost from the start, the child didn't like that, cried after eating it, had diarrhea, and then broke out." A careful history is of even more value than skin tests.

Testing the skin to common foods is of value. It is a laboratory procedure, and one must not depend on it too much. If one has the good fortune to obtain a positive skin test, it means the child is or was sensitive to that food, and such a sensitivity may be extremely significant.

Of greatest value is the use of elimination diets. They were first brought out by Rowe,[3] and have been expanded and changed by investigators since that time. Thus, a child with severe eczema, on whom no testing can be done, can be placed on an almost synthetic diet. For milk, one can substitute a soy-bean preparation — Sobee (Mead-Johnson) or Mull-soy (Borden) — that is perfectly adequate. Crystalline vitamins A and D may be given, and corn meal or rice as a cereal. When the child improves on this regimen, a new food may be added at three-day intervals. As one continues with additions to the diet, there may be a recurrence of eczema. When this happens the last food added is eliminated. With painstaking care, it is quite possible to determine to which foods a child is sensitive and to eliminate them or desensitize him to them. If a patient is found to be sensitive to an important food, oral desensitization is readily accomplished.

The person with allergic eczema has the ability to become easily sensitized, and if he is not treated vigorously in early life, multiple sensitivities occur. The allergic infant with eczema, at least for the first year, is probably sensitive only to foods, and usually not to very many. After the first year, he acquires sensitivities to other foods and substances, such as wool and silk and bacteria. He is always scratching his skin so that it is chronically inflamed and the patient becomes increasingly sensitive, the sensitivity becoming an almost insurmountable problem in adult life.

If one is carrying out a regime like the above only bland ointments are usually necessary. The child is given a starch bath daily, and borated vaseline is applied.

If one prefers not to accept this point of view about allergic eczema, the usual treatment is tar ointment. There are a number of almost colorless tar preparations in greaseless creams containing wetting agents.

Contact Dermatitis

Contact dermatitis is an extremely common inflammation of the skin, due to repeated contact

with normally innocuous substances. This sensitivity is not hereditary and the lesion is generally localized at onset to the exposed areas. Unfortunately, as with the drug eruptions, there is almost no substance to which the skin may not become sensitized; the commonest such substance is poison ivy.

The diagnosis of contact dermatitis is often made through the history. The use of patch or contact tests is very helpful. It is not necessary to have any elaborate setup for this type of testing. The suspected substance should not irritate the normal skin (one can put a patch on himself first, and then try it on the patient). For example, if the patient is thought to be sensitive to turpentine, one can make a 1 per cent solution of turpentine and put it on the normal skin, cover it with a piece of adhesive tape, and leave it on for twenty-four hours. If he is sensitive to it, an erythema with vesicles will appear at the test site.

The treatment depends on the stage of the eruption. If the eruption is acute, wet dressings or baths are indicated. If it is subacute, lotions or pastes are used.

It has been shown that almost anyone can become sensitive to poison ivy. Usually the person who pulls it up and says, "Nothing ever happens to me" is the one who finally gets the worst attack. The best approach to the problem is: first, avoid poison ivy; and, second, if you come in contact with it, do something about it as soon as possible. The chances are that if the sensitizing oil is removed from the skin within four hours, one may not get an attack. The affected area should be thoroughly scrubbed with brown soap and water, rinsed well, and sponged off with alcohol.

If soap and water are not available, the ivy may be neutralized by smearing the skin with the juice of the plantain or jewel weed.

If one is susceptible to ivy and unable to avoid it, desensitization is indicated and is often very effective. It may be accomplished by either the oral or the intramuscular route. For oral desensitization the 2 per cent tincture of the poison-ivy toxin is given, beginning with 1 drop and increasing by 1 drop daily up to 1 teaspoonful. This is continued throughout the season. The intramuscular injection should be preseasonal, and usually four injections at weekly intervals suffice.

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MEDICAL PROGRESS

ABDOMINAL SURGERY

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SINCE the effects of trauma inflicted in warfare is, at the moment, uppermost in our minds, it seems appropriate that this progress report be limited to abdominal injuries. Although reports from previous wars and civilian practice have been helpful in determining the best methods of treatment in certain types of abdominal wounds, the classic one, used as a base line in recent years, is the masterful contribution by Wallace.[1] To him, we owe the careful compilation of data collected from the actual experience of the surgeons working under his direction in World War I. Many previously disputed points regarding the management of abdominal wounds were settled. In the present war, most of our information regarding this subject has come from British sources. This is probably due largely to the breakdown in communications and lack of free exchange of journals, but there is some likelihood that political restriction on such information has played a role.[2]

In this "total war," we are faced not only with the problem of wounded combatants but with that of injured civilians regardless of occupation, age or sex. This type of warfare is unique in the memory of man, as are the agents of destruction used. The complete change in the character of the machines of war in the past twenty-five years has brought about new kinds of wounds along with a type of destruction of life and property hitherto unknown. Almost completely surprised and unprepared for such extensive and varied injuries, Great Britain has met the situation in a most efficient and gratifying manner. To her, we in America owe the opportunity for preparedness,

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even though progress in this direction has, at times, seemed unbearably slow.

The most comprehensive article so far written on the types of abdominal wounds suffered by the British in the present conflict is one by Gordon-Taylor[3]; it was presented by him before the Clinical Congress of the American College of Surgeons in Boston (November 3–7, 1941). In the present report he will be quoted often and liberally, since he has brought to us many important details, particularly those concerning the abdominal injuries resulting from bombing. It is my purpose to stress these civilian casualties, since those of us left at home must gather information in advance of such a catastrophe; also, problems relating to the management of abdominal wounds occurring in civil practice are always important. From various reports from the war zones, and especially from the excellent military manual on abdominal injuries prepared by Storck,[4] we can learn much to help us when dealing with civilian wounds.

Abdominal injuries can be roughly divided into two categories: those produced by blunt force and without penetration of the abdominal wall, and those produced by penetrating objects. Both types may be associated with other injuries, such as fractures, chest wounds, cranial wounds and trauma to the soft parts of the body. In the management of such casualties, one must be on the alert for multiple injuries and be ready to treat them in order of importance, or with two or three teams working at once on different regions. The need for early and adequate treatment of intra-abdominal injuries ranks second only to the control of severe hemorrhage.

Shock occurs following all forms of severe trauma. This condition is of varying degrees and usually appears early, but may occur after some hours have elapsed. Blalock[5] classifies patients suffering from shock in two main groups: those who have bled from injured blood vessels and appear with cold extremities due to vasoconstriction, and those whose blood loss has occurred into the great veins of the body owing to vasodilatation and who may have warm extremities. Both types show restlessness, pallor, air hunger, thirst and lowered blood pressure. A rapid, thready pulse occurs in the first group. Shock is best treated by warmth, sedation with morphine and blood or plasma transfusions according to the extent of hemoconcentration. Massive hemorrhage is the only condition warranting immediate surgical intervention before shock has been effectively treated. In abdominal injuries, this preliminary condition must be handled with the greatest expedition since the time factor is of great importance.

Mulholland[6] points out the cause of death in abdominal trauma. The mortality in a group of cases studied by him was 51 per cent. Of these victims, 43 per cent died of shock, 43 per cent of infection, and 13 per cent of hemorrhage. It appears that future efforts will do much to lower this rate, since both shock and infection are under better control than formerly. Heyd[7] calls attention to the simple objective test of the operative state of a patient under these conditions: that is, the warmth of the body. Doubtless many cases are subjected to surgery before shock has been controlled.

Nonpenetrating Wounds

Nonpenetrating abdominal injuries may result from comparatively slight trauma, such as a fall while at play. Various gradations from this to very severe crushing injuries occur. A new type of injury produced by blast has appeared since civilian bombing has been recorded. This occurs when an explosion takes place in close proximity to the victim without any shell fragment's penetrating the body. Petechial hemorrhages have been described in the brain, lungs and abdominal viscera. Actual perforations of hollow viscera have been reported, as well as thromboses of large blood vessels supplying segments of intestine. Similar injuries have been reported to men in the water from nearby explosions in the sea.[8] Most of the intra-abdominal lesions, however, are produced through considerable impact against the abdominal wall by flying or falling objects or by moving vehicles.

Fatheree[9] has collected from the literature the fact that 60 to 78 per cent of intra-abdominal injuries by blunt force affect the solid organs. This is due to the firmer fixation of these structures as compared with the mobility of the hollow viscera. When one is confronted with the problem of intra-abdominal trauma, it is important to realize the possibility of serious injury and to classify each case as one of minor injury to the parenchymatous organs requiring no operation, a hopeless situation with multiple injuries beyond rescue, obvious injury to a hollow viscus or continued hemorrhage from solid organs or the mesentery, or a borderline case in which there is reason for doubt. Both the last-named types require surgery as soon as shock has been controlled and conditions permit. The sooner a rupture of the intestine or bladder is sutured, the greater is the likelihood of a successful outcome.

Liver injuries are of all gradations. The mild ones may be recognized by the minimal trauma and symptoms. If one could be sure that there were no other viscera involved, many exploratory

laparotomies could be avoided. Hinton[10] pointed out years ago that the bleeding in liver injuries has usually ceased at the time of exploration. It has often been observed that it is unwise to disturb such a clot, and one should be content to remove the extravasated blood and bile from the abdominal cavity by means of a suction tip alone. In large rents that are still bleeding, one must either pack the area with gauze, cauterize the bleeding surfaces or attempt suture. Corriden[11] has reported a case of severe subcapsular rupture of the liver successfully treated by a large transplant of rectus muscle into the sulcus. Grey Turner[12] points out that the mortality in liver injury is high and states that hemorrhage accounts for 60 per cent and sepsis for 40 per cent of the deaths. He describes an ingenious method of suturing a rent in the liver. By first placing a large strand of catgut through the substance on each side of the opening parallel to the edges, he is able to prevent his deep transverse sutures, passed outside the transverse strands, from cutting out. Totten[13] and Estes[14] have called attention to digital compression of the hepatic artery and portal vein during the manipulations necessary for controlling the bleeding from the wound itself. Hawthorne, Oaks and Neese[15] report an interesting case of liver injury manifesting itself by repeated massive hemorrhage through the bile ducts.

In a previous publication, I[16] have cited a fatality from autotransfusion of the blood collected from the abdominal cavity in a patient with ruptured liver. This patient died of anuria, probably through the toxic action of the bile on the kidneys. Since this case was published in 1931, various authors have called attention to the danger of autotransfusion in liver injuries. The term "hepatorenal syndrome" has been used by Orr and Helwig.[17] More recently McCorkle and Howard[18] have reviewed the question of liver damage followed by other complications, and report a case showing transient urinary suppression following laparotomy for acute ruptured liver. The patient was not autotransfused and recovered. Associated low kidney function has long been recognized as a dangerous sequel to operations on patients with chronic liver disease. Coller, in his discussion of Orr and Helwig's paper, suggests that oliguria, under these circumstances, might be attributed to other physiologic alterations so often associated with poor liver function rather than to any specific toxin generated by the damaged liver.

The *spleen* is injured with considerable frequency. This condition may manifest itself by overwhelming shock, pain referred to the shoulder and extreme leukocytosis. On the other hand, many cases of delayed bleeding are reported.[19–22] An injured ballplayer, for instance, may even continue in the game for a while, or may not be suspected of a serious injury until exertion several days later produces a second burst of bleeding. McIndoe[19] collected a total of 45 cases of bleeding delayed from two days to six months after the initial trauma. He classifies spleen injuries as follows: minor, superficial, capsular rupture or slight splenic contusion producing parenchymal ecchymosis; intrasplenic hematoma and subcapsular hemorrhage without capsular rupture; and capsular and parenchymal rupture with pericapsular hematoma. Rousselot[23] adds avulsion of the spleen from the hilus vessels. He calls attention to the over-all mortality of 30 per cent in injuries to the spleen, but in his group of 9 uncomplicated injuries, all the patients recovered. He made follow-up studies of these cases in order to determine that late secondary anemia does not occur following splenectomy. All authorities admit that splenectomy is the treatment of choice. Few advocate suture or tamponade. One known V-shaped rent in the spleen, discovered during laparotomy for a more severe injury, was left untreated and recovery took place.[24] Multiple complicating injuries and delayed improper treatment account for the high mortality.

Kidney injuries without penetration are of common occurrence. More of these of severe proportions have occurred since the high-speed automobile was developed. Authorities are somewhat divided regarding early surgery versus conservative measures. Nonoperative treatment certainly results in a successful outcome in the majority of kidney contusions. On the other hand, there are valid reasons for removing hematomas about the kidney.[25] All agree that one must determine the presence of a functioning kidney on the opposite side. Intravenous pyelography is the method of choice, although some authorities prefer retrograde pyelography, since this gives a better idea of the extent of the injury. Peacock[26] reports a fatal hemorrhage following retrograde pyelography for ruptured kidney. Robertson[27] and others stress conservative surgery when any is done. Turton[28] gives experimental data to support repair of the kidney. He also advises exploration through a loin incision except in cases with complicating intraperitoneal lesions. His dictum is, First, save the patient, then save the kidney. When the renal pedicle is torn, a rapidly developing mass in the flank will manifest itself; under these circumstances, nephrectomy is justifiable and usually necessary. Associated injuries to the descending colon require laparotomy. Cases demanding operation are naturally associated with a higher mortality than are those of less severe trauma.

The *pancreas* may be injured by blunt force, owing to its fixed position across the ridge of the spine. Few cases of successful immediate operation are reported. Cysts may form, which can later be removed or marsupialized.[29] Dixon[30] reports 2 cases of pancreatitis associated with rupture of the duodenum. There was no evidence of injury to the pancreas in either case. Both patients died and one was studied at autopsy. The condition cited implies a mystifying mechanism, since acute perforation of the duodenum from ulcer is rarely if ever associated with pancreatitis.

Hollow viscera, when ruptured, almost invariably result in death unless prompt action is taken to remedy the defect. The earlier operation can be undertaken, the better is the opportunity for a successful outcome. One must often explore the abdomen under these circumstances, to rule out a suspected perforation. There is little or no hazard from a negative exploration under modern conditions, and no apologies are needed for this procedure. It is not good judgment to delay exploration until the diagnosis can be confirmed by x-ray films that demonstrate free air in the peritoneal cavity, or to wait until the signs of peritonitis manifest themselves.

Whenever possible, rents in the stomach and intestine should be closed without resection. If the bowel has been stripped of its mesentery or the rent goes completely through the bowel into the mesentery, resection and anastomosis are justifiable. In colon wounds it is best to suture the opening if it is a small one and in a fixed portion of the bowel. If it is in the free mobile cecum or sigmoid, exteriorization is the method of choice. In perforations of the colon not exteriorized, a proximal vent should be established after suture. One must have in mind the possibility of traumatic thromboses of the large blood vessels, as such cases have been caused by blast injuries.[2] In the event of doubt concerning the viability of a segment of bowel, it is best to do a proximal complete colostomy.

It may be irrelevant to mention at this point injuries to the colon from compressed air. It seems, however, that this hazardous prank cannot be overemphasized. Brown and Dwinelle[31] collected 60 such cases from the literature and added 3 of their own. It is interesting to note that as little as 4 pounds of pressure can cause rupture of the colon. The use of compressed-air hoses for dusting off workmen's clothing in industrial plants should be forbidden. The mortality in these injuries is 100 per cent without surgery and 42 per cent with it.

The *urinary bladder* may be ruptured by blunt force. Damage to this organ should be looked for in all traumas to the low abdomen. Injury to the urethra and bladder are often associated with fractures of the pelvic bones. The simplest method of diagnosis is to catheterize the patient, if he cannot void. A large amount of bloody urine indicates a kidney lesion. Absence of urine or a few drops of bloody urine indicate bladder injury. Weyrauch and Peterfy[32] have shown that cystometrogram tests are unreliable as a means of diagnosing a ruptured bladder. Peacock[33] reports 28 cases with only 4 deaths. Eighty-five per cent of his cases were associated with fracture of the pelvis. Early recognition and early operation were responsible for his good results.

Penetrating Wounds

Penetrating wounds of the abdomen require less diagnostic acumen than nonpenetrating ones. If a large vessel is severed, death ensues so rapidly that the patient does not live to reach the surgeon. Thus, patients with lateral anteroposterior wounds are the ones he generally sees, whereas those with wounds in the midline seldom come to him before death. Gordon-Taylor[3, 34, 35] points out that wounds penetrating the abdomen from one side to the other are more serious than those proceeding anteroposteriorly. Also, he calls attention to the combination of abdominal and chest wounds. Apparently, the left hypochondrium is more vulnerable than any other quadrant. Thoracic surgeons have developed the chest approach to this region — many of them prefer to attack the spleen and the upper end of the stomach by this route. Multiple injuries are as a rule more dangerous than single wounds; much depends, however, on the extent of the injury. The degree of hemorrhage is, according to Storck,[36] the most important factor in success or failure.

One should make a definite study of the course of the projectile. If there is only a wound of entry, a single film will locate the foreign body. It is not essential to remove it in all cases, but one should know in what region it lies. This gives the best clue concerning the organs most probably injured. Great stress is placed on buttock wounds, since they so often range upward. The fragment of shell casing or bullet may well lodge in the urethra or bladder, penetrate the rectum or traverse the abdomen. The most serious of all omissions is to overlook an injury to the colon, since recovery is extremely rare if perforation of this viscus is not recognized and properly treated.

In penetrating injuries of the abdomen, regardless of the direction of the projectile or its size, one must at the earliest possible moment determine by operation the extent of the injury and do the logical repair of the wounded parts. If there turns out to be only a superficial tract, traversing portions

of the abdominal wall, so much the better. Perforations of the stomach must be expected to have a wound of exit on the posterior wall. One should approach the deeper wound by dividing the gastrocolic omentum or by anterior gastrotomy. Wounds of the duodenum may involve both walls. Fraser[24] cites a case of complete severance of the duodenum treated successfully by turning in both ends and doing a posterior gastrojejunostomy. Perforations of the small intestine should be sutured without resection wherever possible. The mortality is lower in wounds of the jejunum than in those of the ileum.[37] After operations on the small intestine for traumatic wounds, one should employ Wangensteen suction or, if possible, suction drainage by a Miller–Abbott tube or some modification of it.

The colon presents a more serious problem; many of the pertinent points regarding this structure have already been made in the section on nonpenetrating wounds. Ogilvie,[38] of the British Army, emphasizes the importance of exteriorization when possible and proximal colostomy if suture is accomplished. These are good general surgical principles and should be rigidly observed. Drainage to the site of injury is advocated, care being used to keep the end of the drain away from the suture line.

Chemotherapy is rapidly becoming standardized. The tendency to apply the sulfonamide drugs locally is now being seriously questioned. Kennedy[39] states that all combatants are furnished with some form of these drugs, to be taken by mouth, if possible, as soon as a wound is received. Doubtless it will be possible to give many of the injured sulfadiazine by the intravenous route at an early hour. This method was found of inestimable value in treating a large series of severe burns in a recent civilian catastrophe.[40] It seems probable that the local use of the drug will be reserved for emergency dressings or for its bacteriostatic effect in the treatment of contaminated wounds. The sulfadiazine level in the blister fluid of a burn is comparable to that in the blood stream.[41] There is a widespread use of sulfanilamide powder intraperitoneally following any gastric or intestinal operation. There is a rapid absorption of this drug through the portal system. Lyons[42] has pointed out that this may, under certain circumstances, be very dangerous to the liver. The greatest of all sins is to depend on this drug to offset faulty technic.

It appears that the secondarily invading or late developing staphylococcus, responsible for so many deaths and such prolonged sepsis and disability, may be curtailed or even eliminated by the use of penicillin.

Anesthesia in abdominal trauma is a controversial subject. Local anesthesia is advocated by very few. Spinal anesthesia is preferred by Storck[36] and Bove,[43] and Pentothal Sodium intravenously by Ross and Hulbert[44]; the latter was also found useful by Moorhead[45] at Pearl Harbor. Strode[46] and many others conclude that ether is after all the safest and easiest anesthetic for most abdominal wounds.

Wound preparation has been dealt with by Reid and Carter,[47] Koch[48] and Kerrigan.[49] It is obvious that chemicals are of secondary consideration. Careful mechanical cleansing by neutral soap and water with cotton pledgets for ten minutes under sterile precautions obviates the use of antiseptics. The traumatic wound must be kept covered and the washing done away from, rather than toward, it. After this is done, the wound itself is irrigated with warm physiologic saline solution. Reid and Carter prefer to omit the wound irrigation. This is doubtless good advice to those who understand proper wound excision.

First-aid treatment of wounds of the abdomen should consist in the application of a sterile dressing only; this should be held in place by a suitable binder and not be removed until the patient is ready for the operating room. Too much stress cannot be laid on the possible contamination of wounds by ungloved and unmasked personnel prior to definitive treatment.

Ogilvie[38] has called attention to too wide an excision of skin. He says, "Skin is viable after trauma, resistant to infection and irreplaceable — no more must be removed than the bruised edge, that is, one-eighth inch at the most." He further points out that patients with abdominal wounds should not be transported early after operation, but should be allowed to recover from it before being moved any distance. Caution is given concerning the primary suture of traumatic wounds, since too many of them become septic. Delayed closure is advocated. It is important not to pack the wounds too tightly with vaseline gauze, and dressings must be made tight for transportation purposes. Plaster of Paris reinforcement during transportation has been found useful.

Tetanus antitoxin and gas-bacillus antiserum should be used following all civil wounds; the former can be omitted if it can be ascertained that the victim has previously been immunized with tetanus toxoid, but a "booster" injection of toxoid should be administered.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 29041

PRESENTATION OF CASE

A seventy-one-year-old insurance broker entered the hospital because of inability to swallow solids or liquid food.

Three months prior to admission he gradually became unable to swallow solid food, choking whenever he attempted to ingest anything other than fluids. He then developed a similar trouble in swallowing liquids. Whenever he drank liquids he complained of pain and burning in the throat. The patient attributed his trouble to infected teeth, which were extracted on the day prior to admission. During his illness he lost approximately 40 pounds.

The family and past histories were noncontributory.

Physical examination revealed a markedly emaciated, weak, pale man who was having considerable difficulty because he seemed to be choking on mucus collected in the mouth and throat. At times he seemed slightly confused. The skin was thin, white and atrophic. The pupils were equal, round and reacted to light. There was a slight nystagmus on extreme deviation to the right or left, and a senile ectopion. The patient was edentulous, but there were several draining tooth sockets. The tongue was red, smooth and fissured. The pharynx was congested. Skin fissures were prominent at the angles of the mouth. The trachea was in the midline. Expansion of the chest was fair. There was impairment in resonance at the left apex anteriorly, and breath sounds were slightly suppressed at the right base posteriorly. No rales were audible. Cardiac dullness was percussed 8 cm. to the left of the midsternal line in the fifth intercostal space. The cardiac rhythm was totally irregular, but no pulse deficit was noted. A systolic murmur was audible at the apex. The abdomen was held tense, and there was a questionable generalized abdominal tenderness. There was pitting edema of the legs, feet and sacrum.

The blood pressure was 155 systolic, 60 diastolic. The temperature was 100°F., the pulse 68, and the respirations 20.

Examination of the blood revealed a hemoglobin of 8.9 gm. per cent, a red-cell count of 2,970,000 and a white-cell count of 11,200, with 47 per cent polymorphonuclear leukocytes, 7 per cent large lymphocytes, 29 per cent small lymphocytes, 14 per cent monocytes, 2 per cent unclassified cells and 1 per cent nucleated red cells. In the smear, there was a moderate variation in the size of the red cells; many were larger than normal, and rare microcytes were present. There was a moderate achromia, and the platelets were decreased slightly.

On the second day a barium meal was attempted. On fluoroscopic examination of the esophagus, the pharyngeal muscles appeared to be paralyzed. There was pooling of the barium in the hypopharynx, and the barium passed upward into the nasopharynx. The cervical esophagus was narrowed, and it was difficult to visualize this area because of the patient's weakness and the small amount of barium that passed through this area of narrowing. There was evidence of pressure on the esophagus by the thyroid gland at the level of the sternoclavicular joint, and the esophagus was displaced to the right. A small amount of barium spilled over into the bronchial tree.

When the patient returned from the X-ray Department he seemed to be choked up with barium. Suction was instituted, but he died approximately half an hour later.

DIFFERENTIAL DIAGNOSIS

DR. JACOB LERMAN: There is no statement about orthopnea or distention of the neck veins. The trachea was in the midline, and there were no palpable lymph nodes. The thyroid gland was not felt. Was the patient resting quietly lying down, or was he propped up in bed?

DR. TRACY B. MALLORY: There is no statement regarding orthopnea.

DR. LAURENCE L. ROBBINS: I did the fluoroscopy. The patient was not in particularly good condition, but so far as I could determine in the dark room he seemed like a good many patients who have considerable difficulty in breathing and swallowing at the time of examination. His condition did not seem particularly alarming; however, we did not give a great deal of barium. With the first mouthful of barium it was noted that there was widening of the hypopharynx and that the pyriform sinuses were completely filled. A small amount of barium spilled into the esophagus, but it seemed to be more from gravity than from muscular action; it was seen that the cervical esophagus was somewhat narrow. Whether there was actual intrinsic tumor there, I could not be positive, but on the spot films the mucosa looks pretty good. There is evidence of narrowing of the esophagus at the level of the sternoclavicular joint, and it is displaced considerably to the right side. On

this film you can see that the barium has spilled back into the nasopharynx, which is good evidence of paralysis of the pharyngeal and palate muscles.

DR. LERMAN: What about the trachea?

DR. ROBBINS: It is normal so far as I can see.

DR. LERMAN: Why did you say the thyroid gland was the cause of pressure?

DR. ROBBINS: Because of the location of the lesion. I cannot be sure, however, that it was not something else. On this film you can see flecks of barium that are in the right main bronchus, and some of them are even as low as the right-lower-lobe bronchus.

DR. LERMAN: We have a markedly emaciated old man who had considerable difficulty in swallowing, an obvious nutritional deficiency, a moderate anemia of the normochromic, slightly macrocytic type and x-ray evidence of pressure on the esophagus, with displacement toward the right. From the story I gather that the deficiency syndrome antedated the dysphagia. It is difficult for me to be certain of that. Could he have had deficiency of this degree after only three months of esophageal obstruction?

Let us consider the thyroid gland as the cause of his trouble. It is hard to believe that any thyroid tumor could compress the esophagus and displace it to this degree without involving the trachea. Certainly thyroid malignancy that could obstruct the esophagus almost completely would do so only terminally, at least according to my experience. Chronic thyroiditis can obstruct the esophagus by posterior extension of the process, but I do not see how it can displace the esophagus. Moreover, I have never seen chronic thyroiditis produce almost complete obstruction. A toxic nodular goiter that is substernal, such as this would have to have been, should be considered. It would at least explain the relatively rapid development of the deficiency syndrome. In cases of thyroid disease, if there is clinical evidence of one or another deficiency syndrome, one must suspect associated hyperthyroidism. I have seen several thyrotoxic patients with peripheral neuritis and other evidence of vitamin B deficiency. At the present time I have under my care a patient with hyperthyroidism and marked dysphagia, associated with glossitis and pharyngitis of the type seen in vitamin B deficiency, whose basal metabolic rate is +55 per cent. She has been on a poor diet because of anorexia more than anything else. I cannot imagine a thyroid nodule that could produce this much pressure on the esophagus without producing pressure on the trachea and other evidence of pressure in the superior mediastinum. Obviously there were no symptoms of hyperthyroidism. Therefore it seems to me that we have to discard toxic goiter as a cause of the esophageal obstruction. On the other hand there should have been found at autopsy an ordinary nontoxic nodule, which was independent of the main cause of death.

Lymphoma has to be considered, but there is nothing specific in the record to lead to this diagnosis.

I shall now consider two diseases. Generalized deficiency, no matter how it develops, may produce the picture of Plummer–Vinson syndrome, which this patient may have had. However, it is usually associated with hypochromic anemia. I believe that Plummer–Vinson's disease is part of a vitamin B deficiency syndrome, and that this case represents one type. Many of these patients with deficiency are prone to develop malignancy of the mucosa — mouth, pharynx or esophagus. Consequently it is likely that there is malignancy in the upper part of the esophagus in this case. One can reason the other way around: he had esophageal malignancy to start with and the interference with swallowing of food caused development of the deficiency state. Against this course is the relatively short duration of the obstructive symptoms.

In addition, there is the cardiac picture, which was probably due to arteriosclerotic heart disease with auricular fibrillation and, possibly, congestive failure. Other than peripheral edema, there was no evidence of congestive failure. One might therefore suspect that the edema was due to deficiency disease (nutritional edema) and not to congestive failure. Thus I am left with the following diagnoses: carcinoma of the esophagus and nutritional deficiency, with an independent, nontoxic, nodular substernal goiter, and arteriosclerotic heart disease.

DR. FULLER ALBRIGHT: Is it unusual to have the esophagus displaced by carcinoma?

DR. LERMAN: It depends on where the tumor arises. Most malignancies of the thyroid are asymmetrical.

DR. ALBRIGHT: It would constrict it down but would not displace it?

DR. LERMAN: I cannot answer that.

DR. MALLORY: I can imagine displacement, Dr. Albright, especially if the tumor extended into the deep cervical nodes on one side of the neck more extensively than on the other.

DR. EDWARD B. BENEDICT: This is not the usual picture of carcinoma of the esophagus. To have paralysis of the muscles of the pharynx is not usual either; I have never seen that except in myasthenia gravis.

DR. ROBBINS: It is occasionally attributed to pseudobulbar paralysis. This looks a good deal like the hypopharynx in myasthenia gravis except

that one sees only weakness whereas this looks like nearly complete paralysis.

DR. BENEDICT: You said you thought the mucosa was normal.

DR. ROBBINS: So far as I could tell.

DR. BENEDICT: That would certainly be against carcinoma of the esophagus, if intrinsic, as well as against myasthenia.

DR. J. H. MEANS: How is myasthenia going to push the esophagus to one side?

DR. BENEDICT: That would not do it alone.

DR. ROBBINS: I do not believe that the location of the lesion had anything to do with the paralysis of the pharyngeal muscles, because the mass is too far away from the point where paralysis is most noticeable.

DR. MEANS: That paralysis bothered me no end. I do not see how you can hook it up with the same trouble from the diagnostic point of view.

DR. MALLORY: I might just as well confess that we are not going to explain the paralysis.

CLINICAL DIAGNOSIS

Carcinoma of esophagus.

DR. LERMAN'S DIAGNOSES

Carcinoma of esophagus.
Nutritional deficiency.
Nontoxic nodular goiter (substernal).
Arteriosclerotic heart disease.

ANATOMICAL DIAGNOSES

Substernal colloid goiter.
Benign ulcer of esophagus.
Arteriosclerosis, moderate: aortic, coronary and cerebral.
Pulmonary emphysema.
Emaciation.

PATHOLOGICAL DISCUSSION

DR MALLORY: This man had a very large substernal goiter, weighing 350 gm., which clearly was the cause of the displacement of the esophagus. He also had a benign ulcer of the esophagus at a much higher level; it was only 2.5 cm. below the epiglottis, too high to be in any way connected with the thyroid tumor itself. We have no explanation, as I said, for the pharyngeal paralysis. The possibility of peripheral neuritis did not occur to us and was not called to our attention at the time of autopsy. We did go through the cerebrum and medulla with some care, and there was no medullary lesion to account for the paralysis. I cannot eliminate the possibility of peripheral paralysis, although we have no evidence that other peripheral nerves were involved.

DR. LERMAN: The Plummer–Vinson syndrome has been described several times in association with paralysis of the pharynx. That is why I attributed the paralysis to the deficiency state.

DR. BENEDICT: What was the cause of death?

DR. MALLORY: There was extensive emphysema. There was mild coronary sclerosis without occlusion, and marked emaciation, which was noted clinically; we found nothing else.

DR. BENEDICT: He apparently did not inhale much barium.

DR. MALLORY: Traces of barium were found down to the end of the bronchial tree. On the other hand as much and more lipiodol is frequently instilled without any respiratory embarrassment. I am sure that the patient did not die of bronchial obstruction.

DR. ROBBINS: Many of the patients who have paralysis of the hypopharynx spill barium into the trachea in larger amounts than this man did without any sequelae.

CASE 29042

PRESENTATION OF CASE

A sixty-six-year-old plumber entered the hospital because of shortness of breath.

For many years he had had a chronic hacking cough that occasionally was productive of small amounts of sputum. Six months before entry, he had to give up working because of generalized weakness, and four months later he became short of breath after only moderate exertion. The dyspnea became progressively worse, and he became orthopneic. Four weeks before entry his cough increased and he raised several teaspoonfuls of sputum, which was pink stained at times. During the month prior to admission his arms, neck and jowls became swollen, particularly after lying down, and veins appeared over his chest. At no time did his ankles swell. His appetite was unchanged, and his weight had been maintained at 137 pounds.

The family history was noncontributory. Fourteen years before entry he was found to have a systolic blood pressure above 200. He never noted any frequency, urgency, hematuria, pyuria, nocturia, dysuria, costovertebral-angle tenderness or headache.

Physical examination revealed a well-developed man who was obviously dyspneic and orthopneic. There was a striking pitting edema of the upper extremities, and engorgement of the veins of the upper extremities and neck. There were many dilated veins over the chest to the level of the costal margins. There was slight cyanosis of the

lips. Many small matted lymph nodes were felt in the left supraclavicular fossa and left axilla. The trachea was deviated to the right. Over the right lung anteriorly and posteriorly there were greatly diminished breath sounds. There was no change in the transmission of fremitus or spoken voice. There was dullness at the right lung base posteriorly, and diaphragmatic excursions could not be demonstrated on that side. The cardiac dullness was percussed 7.5 cm. to the left of the midsternal line. The heart sounds were of good quality, and no murmurs were audible. The liver was percussed 5 cm. below the costal margin. The spleen was not felt. There was no edema of the lower extremities.

The blood pressure was 210 systolic, and 100 diastolic. The temperature was 98.8°F., the pulse 80, and the respirations 20.

Examination of the blood revealed a hemoglobin of 10.2 gm., a red-cell count of 3,200,000, and a white-cell count of 14,100, with 83 per cent polymorphonuclear leukocytes, 7 per cent large lymphocytes, 3 per cent small lymphocytes, 3 per cent monocytes, and 4 per cent myelocytes; there was a slight anisocytosis of the red cells. The urine was acid, had a specific gravity of 1.018, gave a ++ test for albumin, and contained numerous hyaline and finely granular casts. The Sulkowitch test for urinary calcium was ++. The blood nonprotein nitrogen was 28 mg. per 100 cc.; the bicarbonate was 25.3 milliequiv., and the chloride 86.4 milliequiv. per liter.

A chest roentgenogram demonstrated a lobulated mass in the right upper mediastinum apparently arising from the tracheobronchial angle, with several smaller masses lateral to it. The heart was slightly displaced to the right and was slightly enlarged. The diaphragm was low on both sides, the right half showing impaired motion. The right lower lobe of the lung was decreased in size, as visible in the lateral view, and contained flecky areas of increased density. The soft tissues of the neck were enlarged, particularly on the right, and there were several areas of calcification on both sides. Venous-pressure studies were done with the patient sitting at a 60° angle with the bed. The pressure in the right antecubital vein, held at the level of the second costochrondral junction, varied from 400 to 410 mm. of water (normal, 40–110 mm.) in three determinations, and showed a good pulse excursion. The simultaneous pressure in a vein over the internal malleolus was 100 mm. A biopsy specimen obtained from the left axilla showed only chronic inflammation. A bronchoscopy was suggested, but it was thought that the patient was too sick to withstand the procedure.

On the eighth day 150 r of x-ray therapy was given to the mediastinal mass. That afternoon he was found to be unresponsive. The entire body was flaccid. The pulse was 90, and the respirations 20. He was in an oxygen tent at that time. Two hours later he died. Terminally the respirations varied between 35 and 25.

Differential Diagnosis

Dr. Austin Brues: May we see the x-ray films?

Dr. Laurence L. Robbins: This is the lobulated mass that was described, with these other areas of density lateral to it; I note that the right lower lung field is less bright than the left. In the lateral view you can see that there are small areas of increased density posteriorly, so that the pulmonary lesion must be located in the lower lobe. The areas of calcification that were described in the neck are these. I think the diaphragm is low on the left, but that does not agree with the record, which states it was low on both sides.

Dr. Brues: It is helpful to have the right side high as well as paralyzed.

That is not a filling defect in the stomach, is it?

Dr. Robbins: No; I do not believe so.

Dr. Brues: The differential diagnosis is that of upper mediastinal obstruction with compression of the vena cava, presumably including or below the azygos vein because of the venous distention and massive edema. Aside from that we have nothing but an enlarged liver and hypertensive renal disease, to which I shall refer only in the final diagnosis. Mediastinal compression can be due to tumor, benign or malignant,—infection or aneurysm. Taking up the possibilities, carcinoma of the lung, arising in the tracheobronchial angle, with metastases to the bronchial nodes would account for the greater part of the picture. I think the lesion and metastases were suitably located for this. We should have to include a lesion below, either a metastasis or a plugged bronchus, to account for atelectasis in that region—I take it that the lower lobe was atelectatic. Against the diagnosis of carcinoma of the lung we should consider the long history of cough. The history does not say for exactly how many years he had been coughing. It says "many years." I believe if that meant a year and a half or two years it would be consistent with cancer. Otherwise, we have to assume he was coughing for some other reason before the present illness. With calcification in the neck he probably at one time had tuberculosis of the cervical nodes, but we have no evidence of old pulmonary tuberculosis. The absence of weight loss is against carcinoma, but weight loss is certainly not always found in cancer. Hemoptysis is one of the early

symptoms indicating erosion of the bronchus and is therefore in favor of cancer.

This exact picture may also be produced by tuberculosis of the bronchial nodes; chronic tuberculosis of the mediastinum is common in children but extremely uncommon in adults, and therefore is very improbable. As for Hodgkin's disease, lymphosarcoma or leukemia, hemoptysis is not usually an accompaniment of these conditions. The localization of the mediastinal involvement to the upper mediastinum is somewhat more characteristic of carcinoma. Certainly, I should say that lymphoma or some other radiosensitive tumor is by no means out of the question. It is my second best diagnosis, and I think it was perfectly correct that radiotherapy in lymphoma doses was given; this should always be done in such a case if the diagnosis cannot be settled.

We must include in the differential diagnosis of mediastinal tumors, such conditions as benign tumors, teratomas and localized infections. These would be expected to displace things away from the lesion because they grow by expansion rather than by infiltration. We might consider aneurysm, but there is nothing to suggest it. There is no serologic report, but I should not go all out in favor of aneurysm even if the Hinton test were positive.

As for metastatic malignancy — it is anybody's guess where the malignancy might be. Perhaps an intra-abdominal tumor would be most likely to do this. If this is carcinoma of the lung, which I think on the basis of the whole picture is the best available diagnosis, there are metastases to the bronchial nodes. Most statistics show that the majority of carcinomas of the lung metastasize to the regional nodes and that the next commonest site is the liver. With an enlarged liver, we might therefore assume it to be involved. What about involvement of the adrenal glands? The chloride was low (86.4 milliequiv.), the blood having presumably been taken from the antecubital vein in the edematous area. There was obvious venous stasis. Under these circumstances one would expect the chloride to be low because of selective diffusion out of the bloodstream into the swollen interstitial space. I am not going to believe that the chemical findings indicate anything else, although bilateral adrenal metastases are frequently seen in cancer of the lung.

My diagnosis therefore is carcinoma of the lung arising in the right bronchus, with metastases to the bronchial nodes, and involving the vena cava and azygos vein by pressure or thrombosis and probably the phrenic nerve, since the paralyzed diaphragm is high and not low. I should say that there was partial atelectasis of the lower lobe due to bronchial obstruction or compression from metastases, metastases to the liver but probably not to the adrenal glands; hypertensive heart disease, with slight vascular changes in the kidneys; and, finally, old tuberculous infection in the cervical nodes.

Dr. J. H. Means: I saw this man just before he died. I was not confident that he had lymphoma, but thought he was entitled to treatment. However, there was evidence of such severe mediastinal pressure that we debated about giving it because of the question of acutely increasing the pressure and perhaps killing him. But finally, after consultation with the X-ray Department, it was decided to give a small dose. I talked it over with the Surgical Service and raised the question of whether splitting the sternum would offer a feasible method of relieving the upper mediastinal pressure, but they did not believe it advisable.

Dr. Jacob Lerman: When this patient first came in, without having seen the x-ray films but knowing that he had lymph nodes in the axilla and one very large one in the sternoclavicular region, I believed that he had lymphoma. After the x-ray examination the tendency was to swing away from this diagnosis toward carcinoma, either in the mediastinum or in the bronchus.

Dr. Rulon W. Rawson: I saw this patient in the Out Patient Department. At that time he presented no lymph nodes, and we did not consider the diagnosis of lymphoma. The spleen was enlarged, and I was able to demonstrate that the liver was also enlarged.

Clinical Diagnosis

Superior mediastinal tumor (? lymphoma or carcinoma).

Dr. Brues's Diagnoses

Carcinoma of right upper bronchus, with metastases to bronchial nodes and liver.
Secondary compression or thrombosis of superior vena cava.
Atelectasis, right lower lobe.
Hypertensive heart disease, with slight renal vascular changes.
Old tuberculosis of cervical nodes.

Anatomical Diagnoses

Bronchiogenic carcinoma, oat-cell type, with extension to mediastinum and lower cervical region.
Invasion of superior vena cava.
Obstruction of thoracic inlet.
Cardiac hypertrophy.
Nephrosclerosis.
Arteriosclerosis: coronary and aortic.

Pathological Discussion

Dr. Tracy B. Mallory: The autopsy showed a large tumor mass in the upper mediastinum, which encircled the trachea and great vessels. The mass was rather soft and on section was pink and shiny, suggesting sarcoma. However, a quite unusual finding was that the tumor had grown directly through the wall of the superior cava; tumor nodules were even present within the cava itself (actual invasion of the vessel wall by lymphoma is quite unusual). On opening the trachea a number of raised submucosal nodules were felt, and when the right bronchus wall was opened, a mucosal tumor appeared. On the basis that the mucosa of the bronchus was involved and the wall of the vena cava was eroded, we thought at the time of autopsy that cancer was more likely than lymphoma. When the histologic sections came through, the mass turned out to be an undifferentiated small-cell tumor, the cells of which were oval to slightly spindle shaped, so I am quite confident that it was an oat-cell carcinoma of the lung, not lymphoma. We found no distant metastases. There was a small nodule in the adrenal gland, but that turned out to be a cortical adenoma, so that the patient had an excessive rather than a diminished amount of cortical tissue. The liver and spleen were not involved. The deeper nodes of the neck did contain tumor similar to that in the mediastinum. There was also a mass of tumor tissue at the apex of the right lung, which formed sort of a pleural cap over the apex. He had severe emphysema, and I think that his many years of cough were based on that. There was some pulmonary fibrosis, also aspiration of blood into the right lower lobe, which I believe accounts for the density observed by the radiologist in that area.

The kidneys showed mild vascular changes, and the heart was moderately hypertrophied, consistent with some years of hypertension.

Dr. Robbins: Was the tumor of sufficient size to cause obstruction of the right main bronchus?

Dr. Mallory: A very small degree of obstruction only, I should think; there was nothing in the right lower bronchus to produce collapse specifically of the lower lobe as against the upper lobe.

Dr. Edward B. Benedict: Is not bronchiogenic cancer an extremely rare cause of the upper-mediastinum syndrome?

Dr. Mallory: Yes, but we have seen it.

Dr. Benedict: Is it not commoner in lymphoma?

Dr. Mallory: Yes, I think so, although of all mediastinal masses, bronchiogenic carcinoma is the likeliest to extend.

Dr. Means: I should have thought carcinoma more likely to produce such a picture. It encircles the great vessels and squeezes them, whereas lymphoma presses on them.

Dr. Mallory: Lymphoma often invades the mediastinum diffusely. It rarely occludes the arteries, but it closes the veins by external pressure.

Dr. Rawson: But you would not expect lymphoma to invade the vessel wall?

Dr. Mallory: No; it certainly is uncommon.

Dr. Robbins: What did you conclude regarding the immediate cause of death? I am interested because he had the radiation shortly before death.

Dr. Mallory: I think death was due to mediastinal compression, and I do not believe that the radiation made any difference — the patient would have died in approximately the same length of time regardless.

Dr. Robbins: I wondered because of the amount of radiation he had. It was a fairly small amount and not sufficient to cause much swelling of the tumor, particularly in that length of time.

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NATIONAL SOCIAL HYGIENE DAY

National Social Hygiene Day, to be observed this year on February 3, is dedicated to the program for the prevention and cure of venereal disease. Starting with the premise that gonorrhea and syphilis can be prevented and cured, we may well ask ourselves, What has been learned of this prophylaxis and therapy and how may we best utilize our present knowledge?

Adequate education of the layman and the physician must be assured by placing the proper teaching material in the hands of every person competent to use it. Syphilis and gonorrhea must be detected early and treated promptly and prostitution must be suppressed, for the greater the delay the more serious are the complications and the longer the time required for cure. So also must all contacts be sought out and treated and the transmission of disease to future generations must be prevented by premarital examination. Findings from such investigations should be repeatedly confirmed during the early months of pregnancy. Finally, citizens, both physicians and laymen, must co-operate in all phases of this effort.

No magic formulas or secret weapons have been developed, but great advances have been made and are still being made in the treatment of these diseases. The general program of this attack is outlined in the pamphlet "Social Hygiene Takes Battle Stations" issued by the American Social Hygiene Association.

Such a program should be active in every state of the Union. In this connection it is of considerable satisfaction to learn that it is being carried out in far-distant Hawaii* where the venereal disease rate in the armed forces is lower than the average in the Army as a whole. This territory is one of the safest places in the Union from the standpoint of venereal disease among the army personnel. If Hawaii can accomplish this, all other parts of the country can and should do so, especially when we realize how normal living conditions have been disrupted there. The Health Department of Hawaii is to be congratulated on performing a task which Surgeon General Parran has outlined so well as follows: "The conquest of syphilis and gonorrhea is not a task for official health agencies alone, nor yet for physicians alone, but is still a task for the whole people. . . . By the union of public and private efforts we can minimize syphilis and gonorrhea and the ill health, suffering and waste they cause."

*Allison, S. D. Facing the facts about venereal disease. *Hawaii* 3:9, 1942.

EFFECT OF WARTIME FOOD ON CHILDREN IN GREAT BRITAIN

The *Lancet*,[1] editorially, admits that there is as yet little information about child health during the war, and that many factors other than diet may be effective. Thus, it is known that war has resulted in an increased carbohydrate intake for all ages and classes, on account of the shortage of meat, fish,

eggs, milk and fruit, and in a leveling of the diet, since rationing and lack of mass unemployment have brought about a fairer distribution of foodstuffs. The diet of the majority has been impaired, and the diet of the poorest has been improved.

The figures that are available show a definite reduction in the average weight increase in 1940 and 1941 as compared with that in 1936–1939, a rise in infant mortality amounting to 6.5 per thousand live births, and an alarming increase in deaths from tuberculosis. Among children under ten years of age, the deaths from all forms of tuberculosis in 1941 exceeded those of 1939 by 45 per cent, and in children under five, the deaths from tuberculosis of the respiratory tract had nearly doubled. A survey,[2] published in the same issue of the *Lancet*, shows a definite drop, although to no serious degree, in the average hemoglobin level compared with that in prewar groups of school children, and a high incidence of anemia among infants under two years of age. It is admitted that the vitamin C intake is below the optimum, but there is no evidence that this deficiency has retarded the growth or affected the health of British children; doubt has also been cast on the adequacy of the official dosage of "cod-liver oil compound" recommended for infants under six months of age, although no figures exist concerning the incidence of rickets for the winter 1941–1942. Certainly, careful and continued studies are indicated.

On the whole, the diet is considered to be reasonably adequate, although necessarily far from perfect, and is improving; in sharp contrast is the lot of millions of children on the Continent, as observed by Dr. W. D. Robinson in Madrid and by Dr. Harold C. Stuart in unoccupied France and reported by Dr. Stuart[3] to the Suffolk District Medical Society in October. There, with blood ascorbic acid levels invariably zero and low vitamin A levels, many early manifestations of deficiency syndromes were found, and the more recent reports indicate the appearance of even severer deficiencies.

A world on short rations requires very serious contemplation of its nutritional problems.

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MEDICAL EPONYM

Graham Steell Murmur

This was described by Graham Steell (b. 1851) assistant physician to the Manchester Royal Infirmary, in the *Medical Chronicle* (Manchester) (**9**: 182–188, 1888) under the title, "The Murmur of High-Pressure in the Pulmonary Artery."

> I wish to plead for the admission among the recognised auscultatory signs of disease *of a murmur due to pulmonary regurgitation, occurring independently of disease or deformity of the valves, and as the result of long-continued excess of blood pressure in the pulmonary artery.*
>
> In cases of mitral obstruction there is occasionally heard over the pulmonary area (the sternal extremity of the third left costal cartilage), and below this region, for the distance of an inch or two along the left border of the sternum, and rarely over the lowest part of the bone itself, a soft blowing diastolic murmur immediately following, or, more exactly, running off from the accentuated second sound, while the usual indications of aortic regurgitation afforded by the pulse and so forth are absent. The maximum intensity of the murmur may be regarded as situated at the sternal end of the third and fourth intercostal spaces. When the second sound is reduplicated, the murmur proceeds from its latter part. That such a murmur as I have described does exist, there can, I think, be no doubt.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

COMMITTEE ON LEGISLATION

Attention of the members of the Massachusetts Medical Society is invited to the following bills which are of interest to the medical profession and are at present before the Legislature.

House 437. An Act Repealing the Provisions of Law Requiring Pre-marital Serological Tests.

Section 1. Section twenty B of chapter two hundred and seven of the General Laws, inserted by section one of chapter six hundred and one of the acts of nineteen hundred and forty-one and as most recently amended by section two of chapter six hundred and ninety-seven of the acts of nineteen hundred and forty-one, is hereby repealed.

Section 2. Section twenty-eight of said chapter two hundred and seven, as amended by section two of said chapter six hundred and one, is hereby further amended by striking out, in the third and fourth lines and in the thirteenth line the words "sixty days" and inserting in place thereof, in each instance, the words: — six months, — so as to read as follows: —

Section 28. On or after the fifth day from the filing of notice of intention of marriage, except as otherwise provided, but not in any event later than six months after

such filing, the clerk or registrar shall deliver to the parties a certificate signed by him, specifying the date when notice was filed with him and all facts relative to the marriage which are required by law to be ascertained and recorded, except those relative to the person by whom the marriage is to be solemnized. Such certificate shall be delivered to the minister or magistrate before whom the marriage is to be contracted, before he proceeds to solemnize the same. If such certificate is not sooner used, it shall be returned to the office issuing it within six months after the date when notice of intention of marriage was filed.

Section 3. Section fifty-seven of said chapter two hundred and seven, as amended by section three of said chapter six hundred and one, is hereby further amended by striking out, in the second and in the fifth and sixth lines, the words "sixty days" and inserting in place thereof, in each instance, the words: — six months, — so as to read as follows: —

Section 57. Whoever performs a ceremony of marriage upon a certificate more than six months after the filing of the notice of intention of marriage as set forth in such certificate and not having used it fails to return it within six months after such filing, to the office issuing the same, shall be punished by a fine of not more than ten dollars.

House 79. Bill providing for the inspection of colleges, universities and medical schools approved for the purposes of medical education and for withdrawal of such approval in certain cases.

This bill was referred to the Committee on Public Health.

House 80. Bill relative to examination for registration as qualified physicians.

This bill was referred to the Committee on Public Health.

House 81. Bill relative to the scope of examinations for registration as qualified physicians.

This bill was referred to the Committee on Public Health.

House 89. Bill relative to certain clinics conducted by the licensed hospitals.

This bill was referred to the Committee on Public Health.

House 98. Bill relative to payment of medical, hospital and other services to old-age assistance recipients.

This bill was referred to the Committee on Pensions.

House 252. Bill further regulating medical services rendered under the workmen's compensation law.

This bill was referred to the Committee on Labor and Industries; the hearing will be held on February 2.

House 352. Bill relative to payment of medical, hospital and other services to dependent children and their parents.

This bill was referred to the Committee on Public Welfare.

Senate 11. Bill to provide for medical and hospital services and medicines for total and permanent disability cases under the workmen's compensation law.

This bill was referred to the Committee on Labor and Industries; the hearing will be held on February 2.

Senate 62. Bill permitting the registration without examination of certain graduate nurses.

This bill was referred to the Committee on Public Health.

Brainard F. Conley, M.D., *Chairman*
Committee on Legislation

COMMITTEE ON MATERNAL WELFARE

Analysis of Causes of Maternal Death in Massachusetts during 1941

Transfusion Reactions

During 1941, 4 deaths were attributable to reactions from incompatible transfusions.

The first case was associated with complete premature separation of the placenta at thirty-five weeks. The patient, who was twenty-nine years old, had had four previous pregnancies. The first went to term; a living child was obtained, and there were no complications. In the second, third and fourth pregnancies, the babies were born prematurely and were jaundiced, all of which suggests erythroblastosis foetalis. During this pregnancy there had been a little bleeding at the thirtieth week. The patient started labor prematurely and entered the hospital with intact membranes and slight bleeding, which continued with each contraction. When fully dilated, a macerated fetus and a completely separated placenta were delivered spontaneously; this was followed by massive hemorrhage and profound shock. An intravenous injection of glucose, to which the patient responded well, was followed by a 350-cc. transfusion of blood from her husband; the typing and cross-matching were done in the usual rapid fashion, but no incubation of the cross-matching was carried out. The transfusion seemed to have little or no effect. Three hours later, the patient's pulse again became very weak; she was given 500 cc. of blood from a registered donor, but the presence of Rh agglutinins and agglutinogens was not determined. The condition of the patient improved for three days; then she again became weak and was given 500 cc. of blood from her brother-in-law, which was again cross-matched in the usual rapid fashion but no test for Rh agglutinins and agglutinogens was done. Immediately following this transfusion the patient had a violent chill. The respiration became rapid, deep and labored; and from then on she went steadily downhill. A large trace of albumin appeared in the urine, and the sediment contained many casts; the nonprotein nitrogen was 300 mg. per 100 cc. Death occurred twenty-four hours later. An autopsy showed "hemolytic infiltration of the uterine wall, amyloid infiltration of both kidneys and spleen, and generalized edema." This death was apparently due to transfusions of blood,

none of which were tested for the presence of Rh factor; this is supported by the fact that the patient's previous three children had presumably suffered from erythroblastosis foetalis.

The second case was that of a woman who had had six previous pregnancies. The first three were normal. In the fourth, the baby died fifteen hours after birth. In the fifth, a cesarean section was performed because of placenta previa; and following the sixth, because of bleeding, a transfusion was given, presumably from a compatible donor. The patient was delivered vaginally, and the baby lived six hours. The baby showed typical signs of erythroblastosis. After the birth of the baby, the uterus was examined internally and a small rupture at the site of the previous cesarean section was found. A hysterectomy was immediately done. The patient was transfused, but an hour later she vomited blood, bled from the abdominal wound and went into profound shock. A total of 1600 cc. of blood was given, but the bleeding continued. The patient became anuric and jaundiced. Bilateral decapsulation of the kidneys was performed on the eleventh day. There was some improvement in the output of urine, but the patient continued to go downhill and died on the fifteenth day after delivery. It seems likely that this death was due to an Rh reaction.

The third case was that of a patient who had had one previous full-term normal delivery. In this pregnancy, the patient is said to have suffered a post-partum hemorrhage. Ten days after delivery, moderate bleeding again occurred, and a transfusion was given. Death occurred shortly thereafter and was probably due to an anaphylatic reaction, possibly involving the Rh factor.

The fourth case was that of a patient who had had a normal convalescence after a low-forceps delivery at term. Three and a half weeks after discharge from the hospital the patient was readmitted because of an abscess in the right breast. This was opened, and a culture showed *Streptococcus haemolyticus* and *Staphylococcus aureus*. A blood transfusion was followed by a severe reaction; a second transfusion was given twenty-four hours later. The patient developed anuria, with nonprotein nitrogen of 80 mg. per 100 cc., and apparently died because she had been transfused with an incompatible blood.

These four cases evidence the seriousness of blood transfusion. It should never be done until it is certain that the blood is truly compatible. Two of these cases show that the Rh factor is the underlying cause of many anaphylactic deaths.

DEATH

HICKEY — JOHN J. HICKEY, M.D., of Peabody, died January 17. He was born in Marblehead and was in his seventy-sixth year. He received his degree from the Harvard Medical School in 1903.

Dr. Hickey was a staff member of the Josiah B. Thomas Hospital and the North Shore Babies Hospital. His memberships included the Massachusetts Medical Society and the American Medical Association.

His widow, three daughters and two brothers survive him.

NEW HAMPSHIRE MEDICAL SOCIETY

DEATHS

CONANT — MARY C. CONANT, M.D., of Lyme, died August 26. She was in her seventy-first year.

Dr. Conant, the daughter of Jonathan J. and Martha (Howard) Conant, was born in Thetford, Vermont, and graduated from the Woman's Medical College of Pennsylvania in 1899. She carried on an institutional practice in Pennsylvania and New York for many years, and moved to Lyme in 1932. She was a member of the New Hampshire Medical Society and the American Medical Association.

She is survived by a brother and three nieces.

DAY — ARTHUR K. DAY, M.D., of Concord, died on December 31, 1942. He was in his eighty-first year.

A native of Dover, he moved to Concord in 1868 and had been a resident since.

He was a graduate of Harvard Medical School in 1889, and for twenty-five years served as attending physician at the Margaret Pillsbury General Hospital in Concord. During the Spanish–American War he served in the United States Army as a first lieutenant and assistant surgeon. Dr. Day was the school physician of Concord for a number of years.

Besides his widow, he is survived by a daughter, Miss Helen D. Day, and two sons, Captain Philip S. Day, U.S.A., and the Rev. Robert B. Day, of Niagara Falls, New York.

FROST — GILMAN D. FROST, M.D., of Hanover, died October 8. He was in his seventy-ninth year.

Dr. Frost, son of Dr. Carlton P. and Eliza (A. DuBois) Frost, was born in Randolph, Vermont, and graduated from Dartmouth Medical School in 1892. He had been a prominent member of the faculty of Dartmouth Medical School, having served as professor of anatomy from 1894 to 1910, as professor of clinical medicine from 1910 to 1937, as secretary and treasurer of the school from 1896 to 1904 and as treasurer from 1904 to 1909. For a number of years he was health officer of Hanover. He was a member of the New Hampshire Medical Society and the American Medical Association.

He is survived by his widow and four children.

HUTCHINSON — HERBERT S. HUTCHINSON, M.D., of Milford, died November 26. He was in his ninety-fourth year, and when he retired from active practice, a month before his death, he was the oldest practicing physician in New Hampshire.

Dr. Hutchinson, son of Stillman S. and Emeline G. (Lull) Hutchinson, was born in Milford in 1849 and graduated from Dartmouth College in 1875 and from Bellevue Medical College in 1880. He joined the New Hampshire Medical Society in 1882, and received the organization's fifty-year medal a decade ago. He was also a member of the American Medical Association.

He is survived by two sons.

MISCELLANY

TUBERCULOSIS AND THE WAR — HERE AND ABROAD

Information from countries in the war area regarding present health conditions is limited and unsatisfactory. In certain European countries extreme hardship is being undergone, owing especially to nutritional inadequacies. Evidences are already appearing that the sharp rise in tuberculosis encountered during the last war may again be expected, and with increasing severity. Although the death rate from tuberculosis in the United States showed its usual decline last year, increasing effort must be made to maintain this favorable situation.

The United States

The world conflict of 1914–1918 for the first time revealed tuberculosis as a major problem. From the early discovery of large numbers of tuberculous troops in the French army to the final assemblage of mortality records of the war years in the civilian population of all countries engaged, it was evident that tuberculosis was exacting a great toll, unrecognized in the wars of previous years. There is every reason to believe, however, that long wars accompanied by privation have always led to an increase in tuberculosis. Crowding, malnutrition, exposure to infection and hardship of every sort have been considered responsible in different degree.

Fortunately, a quarter of a century of research since World War I has led to a better understanding of methods for the control of tuberculosis. Countries fearing the ultimate outbreak of hostilities, through the tense years preceding their final advent, anticipated tuberculosis as a grave menace and prepared accordingly. But in spite of forewarning and preparation, a rise in tuberculosis mortality rates appears already evident. Modern war is total war. Whole populations are engaged, through accelerated industry as well as actual combat.

In World War II, increasing effort is being made in the United States to avoid the induction of soldiers with tuberculosis. Measures ensuring x-ray examination of practically all recruits admitted to the armed forces are in effect.

In the civilian population, precautions are being taken against nutritional deficiency, since it is almost universally believed to have important bearing on the problem of resistance to tuberculosis. However, malnutrition may not be the gravest predisposing factor in a rise of tuberculosis. The acceleration of industry, leading to crowded quarters in industrial districts, brought about by mass migrations to industrial centers, has created another opportunity for widespread infection.

It is evident that a grave menace exists of another worldwide recrudescence of tuberculosis. Its prevention will require vigorous effort against the spread of infection and all measures possible to maintain a high level of resistance to disease. (Long, E. R. *War and tuberculosis. Am. Rev. Tuberc.* **45**:616–636, 1942.)

Britain

To what extent the tubercle bacillus will repeat its former triumph of a generation ago in Britain cannot yet be properly gauged, but it has taken the initiative and the future course of events will be greatly determined by the effort put forward by tuberculosis workers.

Deaths from respiratory tuberculosis increased about 6 per cent the first year of the war, and 10 per cent the second, while the increase in deaths from other forms of tuberculosis was 2.4 per cent the first year, and 17.6 per cent the second.

A considerable amount of infection is evidenced among the general population, particularly children, which means that either the infecting dose is large or the resistance low. Both causes may have operated in the first half of 1941 when the nightly bombing of towns and cities made contact infection probable and frequent. However, if the increase is found to have continued since more normal conditions have prevailed, it will strengthen the idea that there has been a general lowered resistance to infection in children under five. Many factors may have contributed to the lowering of resistance in children, among which are change in diet, nonpasteurized milk, blackout and shelter conditions, and lack of sleep and rest.

Comparing the trend of events during the first three years of the last war and the available figures for World War II, a definite similarity can be traced, although living conditions now are probably more conducive to the spread of tuberculosis. However, there are some marked differences. Tuberculous meningitis has increased sharply, whereas in the corresponding period of the last war it fell almost to the prewar level. A further point of difference is the small variation between the male and female curves.

These are ominous signs, which mean that infection is lurking in hidden places and taking its toll, especially in infant lives, and which emphasize the urgency of means for discovering these nests of infection and the need for their adequate control. (Heaf, F., and Rusby, L. *A further review of tuberculosis in wartime. Tubercle* **23**:107–130, 1942.)

France

Food rationing started throughout France on October 1, 1940, when the following foods were restricted: bread, meat, cheese, fats, sugar, milk, chocolate and milled products. Technically other foods could be obtained, but in reality it was difficult to get them. The results of a survey carried on by the Institut des Recherches d'Hygiene on how different families of Paris were feeding themselves showed a total caloric insufficiency of about a thousand calories daily, a calcium deficiency and a calcium-phosphorus imbalance, and an insufficient intake of Vitamin A.

Undoubtedly morbidity and mortality from tuberculosis have noticeably increased in Paris. The percentage of rapidly developed tuberculosis has gone up in an alarming manner. Comparing the figures of the first six months of 1941 with the corresponding ones in 1939, the mortality from tuberculosis increased 10 per cent.

Four basic diets were prescribed for sick persons in four specific categories. To lessen the ill effects on persons with active tuberculosis and known lesions, a diet was given that corresponded to their general category, plus a supplementary amount consisting of 45 gm. of meat and 15 gm. of fat daily per patient. Despite the precaution indicated, the march of tuberculosis up to October, 1941, had been ominously progressive; thus if the present dietary regime continues and the consequences increase, the problem of tuberculosis in France will be exceedingly grave. (Minoli, R. F. *Food rationing and mortality in Paris, 1940–41. Milbank Mem. Fund Quart.* **20**:213–220, 1942.)

Canada

The fall in the death rate from tuberculosis in Canada, which has been so evident for the past quarter of a

century, has occasioned in some quarters a false sense of security. Nothing could be more unsound or misleading. A disease that kills nearly 6000 of the population, leaves at least 30,000 incapacitated, and costs the country directly at least $8,000,000 annually is still a formidable enemy and a major public-health problem.

If control of tuberculosis is to be maintained in wartime, tuberculosis services must be continued, problems that arise as a result of the war must be attacked and advantage taken of wartime case-finding projects. Case finding has kept ahead of treatment facilities, which have been inadequate, and until both are developed to a greater degree, control of tuberculosis is still hidden in the future.

Two of the most important phases of case-finding services available are for the general practitioners to provide an early diagnosis, since this is still the greatest source of cases, and examination of contacts, the next greatest source.

Two opportunities have presented themselves as a result of the war: the x-ray examination of all recruits for the armed forces and case-finding projects among industrial workers, particularly in war industries. Tuberculosis is two and a half times as great in industry as in the general population. Therefore, the control of tuberculosis is an important phase of industrial hygiene.

Emphasis is being placed on retaining the open case of tuberculosis in sanatoriums. Every patient who leaves against advice represents a weakness in the tuberculosis-control system. The factors involved should be carefully analyzed and every way possible must be sought to remedy conditions in institutions to offset this failure in efficient segregation. (Wherrett, G. J. *The control of tuberculosis in wartime. Canad. Pub. Health J.* 33:438–445, 1942.) — Reprinted from *Tuberculosis Abstracts* (January, 1943).

CORRESPONDENCE

SUSPENSION OF LICENSE

To the Editor: This is to inform you that on January 20, 1943, the Board of Registration in Medicine voted to suspend for one month the license to practice medicine in this Commonwealth of Dr. Louis R. Medverd, 1210 Cambridge Street, Cambridge, Massachusetts.

H. QUIMBY GALLUPE, M.D., *Secretary*
Board of Registration in Medicine

State House
Boston

BOOK REVIEWS

Source Book of Medical History. Compiled, with notes, by Logan Clendening, M.D. 8°, cloth, 685 pp. New York: Paul B. Hoeber, Incorporated, 1942. $10.00.

There have been forthcoming in recent years several medical source-books, but none, however, with quite the range of this one. It aims at the whole of medicine, starting with the Egyptian papyri and ending with Roentgen, so that there is something in it for every man who finds pleasure and profit in reading, — if not *ipsissimis verbis*, the next thing to it, — a first-hand translation of what many important contributors to medical science had to say.

True, neither this nor any other book of the sort can attain perfection in every eye. There will be complaints no doubt that this has been left out which ought to have been included, and that that has been inserted which has left too little space for something else. And, as is always the case, there are authors who suffer from the brevity of their allotted room — men, if you will, who lend themselves ill to cutting. Pasteur is one such.

But it would be ungrateful to complain too much; all in all this is a noteworthy and valuable assemblage of material, a great deal of which is inaccessible elsewhere. To list the distinguished contributors would be a pleasure, but space does not permit. For each, the editor has furnished a brief note to assist in placing man and matter in historical position, and for those who would go farther in any particular direction he has listed references.

This volume also deserves commendation as a piece of bookmaking. But, if a personal note may be hazarded, the price, even in an era of increasing costs, seems a little high. There are no plates to add to the publishing expense, and it is a pity that the cost will be a deterrent to many who would like to see it on their shelves.

Medical Diseases of War. By Sir Arthur Hurst, D.M. (Oxon.), F.R.C.P. With the co-operation of H. W. Barber, M.B. (Cantab.), F.R.C.P.; H. B. F. Dixon, M.D. (Dub.), F.R.C.P.; F. A. Knott, M.D. (Lond.), M.R.C.P.; T. A. Ross, M.D. (Edin.), F.R.C.P.; and Arnold W. Stott, M.A. (Cantab.), F.R.C.P. Second edition. 8°, cloth, 427 pp., with 8 plates. Baltimore: The Williams and Wilkins Company, 1941. $5.50.

This book, a new edition of one first published in 1916, and later republished in 1918, will be found especially helpful to neurologists and to all interested in the neuroses of war. It is largely based on the author's wide experiences in World War I as neurologist to Guy's Hospital, commanding officer of the Seale–Hayne Hospital for Functional Nervous Disorders and consulting physician to the Salonica Army.

When one realizes that almost 6 per cent of 341,025 discharges from the British Army (from 1914 to the end of April, 1918) were due to war neuroses, the importance of recognizing and treating these cases correctly is evident. Hurst's remarks in Chapter I, "Predisposing Causes of War Neuroses," will repay careful reading. Other chapters follow: "Hysterical Symptoms in Soldiers," "Hysterical Contractures," "Hysterical Postures and Gaits," "Hysterical Tremors" and "Hysterical Blindness," with numerous brief case records, which are instructive. Many results and cures were almost miraculous.

Of especial interest is Chapter XVI, "Anxiety Neuroses of War," by T. A. Ross. Here, in particular, psychotherapy, with explanation to the patient, persuasion and re-education, worked wonders. The chapter on "soldier's heart" is excellent; in great measure, it is based on Sir Thomas Lewis's book, published in 1918. The section by H. W. Barber, on skin disease, is of particular value and importance.

Although this volume is supposed to discuss the medical diseases of war, almost half is given over to the neuroses and almost another half to a number of medical diseases, such as trench fever, typhoid and paratyphoid fevers, bacillary dysentery, epidemic jaundice and tetanus, that will probably be of little importance in World War II, because conditions are so different from those in 1914–1918. Epidemics and perhaps pestilence will probably occur, but they are to be expected among the civilian populations.

Of great value at the end of each chapter are references to the published literature. An index also adds to the general usefulness of the volume. It can be recommended to those who are interested in military medicine.

(Notices on page x)

That was not an idle promise—

when, during the halcyon pre-war days, buyers of G-E x-ray and physical therapy apparatus were assured of maintenance and technical service throughout the life of the apparatus. ¶ To make good that promise—despite trying difficulties which the war now imposes—G.E.'s branch offices and service depots continue to function throughout the United States and Canada. ¶ Of course, it isn't possible for the local G-E service man to call as often as heretofore, what with rationed tires and gas and other transportation problems. But he is always available when there is need for service on equipment. In fact, he feels that the most important part of his job these days is to help keep G-E equipment in the best possible operating condition, for the busier-than-ever physicians' offices, clinics, and hospitals in his territory. *He will not let you down!*

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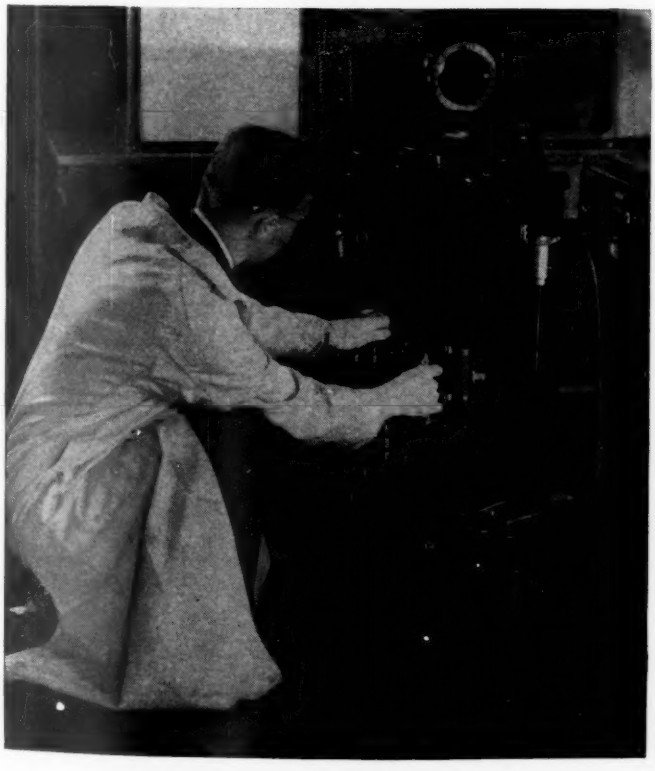

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NOTICES

MIDDLESEX SOUTH DISTRICT MEDICAL SOCIETY

The attention of the members of the Middlesex South District Medical Society is drawn to the fact that the next meeting will be the annual meeting for which the usual notice will be sent.

A special meeting, usually held in January, will not be called for two reasons. The officers of the society feel that there is no special business to be taken up at the present time, and, because of the gasoline shortage, it does not seem advisable to call a meeting for nonessential business.

JOSEPH H. PRATT DIAGNOSTIC HOSPITAL

Bennet Street, Boston
Lecture Hall, 9–10 a.m.

MEDICAL CONFERENCE PROGRAM

Wednesday, February 3 — Prolongation of Action of Subcutaneously Injected Medicines in Man. Dr. Francis F. Foldes.

Friday, February 5 — Psychiatry and the Military Situation. Comdr. A. Warren Stearns.

Wednesday, February 10 — Title to be announced. Dr. John D. Adams.

Friday, February 12 — Some Remarks on the Pharmacology of Digitalis. Dr. L. S. Goodman.

Wednesday, February 17 — Bacterial Allergy. Dr. Ethan Allan Brown.

Friday, February 19 — The Sulfonamides. Dr. C. A. Janeway.

Wednesday, February 24 — Various Effects of Disturbed Androgen-Estrogen Balance. Dr. C. H. Lawrence.

Friday, February 26 — Rheumatoid Arthritis. Major Elam C. Toone, Jr.

On Tuesday and Thursday mornings, from 9 to 10 o'clock, Dr. S. J. Thannhauser will give a medical clinic on hospital cases.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

The monthly clinical conference and meeting of the staff of the New England Hospital for Women and Children will be held on Thursday, February 4, in the classroom of the Nurses' Residence at 7:15 p.m. Dr. Frank Barton will talk on the management of the transfusion service at the Massachusetts Memorial Hospitals (with motion picture). A discussion of the Rh factor will follow. Dr. Jean Black will be chairman.

NEW ENGLAND REGIONAL MEETING, AMERICAN COLLEGE OF PHYSICIANS

Plans for the New England Regional Meeting of the American College of Physicians to be held in the Georgian room of the Hotel Statler, Boston, on February 5, have been announced by Dr. William B. Breed, general chairman of the meeting and governor of the College for Massachusetts. The 1943 annual session of the College has been canceled, and this regional meeting is an official part of the new College program to extend its usefulness and influence during the war.

Clinical sessions will be held from 10 to 12, and from 2 to 5. Dr. Reginald Fitz will act as presiding officer in the morning. and Dr. J. Howard Means in the afternoon. Luncheon will be served at noon. The program will be concluded with a dinner at 6 p.m., following which there will be brief addresses by Rear Admiral Ross T.

Pause at the familiar red cooler for ice-cold Coca-Cola. Its life, sparkle and delicious taste will give you the real meaning of *refreshment.*

McIntire, U.S.N., Major General James C. Magee, U.S.A., Brigadier General David N. W. Grant, U.S.A., Brigadier General Hugh J. Morgan, U.S.A., and Dr. James E. Paullin of Atlanta, Georgia.

All fellows and associates in the New England region are urged to attend. It is expected that the medical personnel of all New England installations of the Army and Navy will be well represented.

MASSACHUSETTS PHYSIOTHERAPY ASSOCIATION

The monthly meeting of the Massachusetts Physiotherapy Association will be held on Tuesday, February 2, at 8:00 p.m., at the Women's Educational and Industrial Union, 264 Boylston Street, Boston. Dr. Francis M. Forster will speak on the subject, "Personality Problems of Handicapped Patients."

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Sunday, January 31

Sunday, January 31

*3:00 p.m. The Application of Psychiatry to Military Needs. Commander A. Warren Stearns (MC), U.S.N.R. The Application of Psychology to the Selection and Maintenance of Aircraft Pilots. Lieutenant Edward J. Galway, U.S.N.R. Symposium on Aviation Medicine. Cheever Amphitheater, Dowling Building, Boston City Hospital.

†4:00 p.m. Common Colds and Their Complications. Dr. Maxwell Finland. Amphitheater of the Peter Bent Brigham Hospital.

Monday, February 1

*12:15–1:15 p.m. Clinicopathological conference. Peter Bent Brigham Hospital amphitheater.

Tuesday, February 2

*9:00–10:00 a.m. Medical clinic. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

*12:15–1:15 p.m. Clinicopathological conference. Peter Bent Brigham Hospital amphitheater.

8:00 p.m. Personality Problems of Handicapped Patients. Dr. Francis M. Forster. Massachusetts Physiotherapy Association.

8:15 p.m. The Prevention and Treatment of Acute Infectious Diseases. Dr. Conrad Wesselhoeft. Greater Boston Medical Society. Beth Israel Hospital.

Wednesday, February 3

*9:00–10:00 a.m. Prolongation of Action of Subcutaneously Injected Medicine in Man. Dr. Francis F. Foldes. Joseph H. Pratt Diagnostic Hospital.

*12:00 m. Clinicopathological conference. Children's Hospital.

Thursday, February 4

*9:00–10:00 a.m. Medical clinic. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

7:15 p.m. New England Hospital for Women and Children. Monthly clinical conference and meeting of the staff.

Friday, February 5

New England Regional Meeting, American College of Physicians. Hotel Statler, Boston.

*9:00–10:00 a.m. Psychiatry and the Military Situation. Commander A. Warren Stearns. Joseph H. Pratt Diagnostic Hospital.

Saturday, February 6

*10:00–11:30 a.m. Medical staff rounds. Peter Bent Brigham Hospital.

*Open to the medical profession.

†Open to the public.

February 3–26. Joseph H. Pratt Diagnostic Hospital, medical conference program. Page x.

February 10. New England Oto-Laryngological Society. Massachusetts Eye and Ear Infirmary.

February 13. American Board of Obstetrics and Gynecology. Page 690, issue of October 29.

District Societies

BRISTOL SOUTH

May 6. New Bedford.

ESSEX NORTH

May 5.

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*A New Type of Medication to Be Used in Bronchial Asthma and Other Allergic Conditions. *New England Journal of Medicine* 223:843–846, 1940.

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ESSEX SOUTH
- February 3. Lynn Hospital.
- March 3. Essex Sanatorium.
- April 7. Addison Gilbert Hospital, Gloucester.
- May 5. Salem Country Club.

FRANKLIN
- March 9.
- May 11. Annual meeting.

All meetings are held at 11:00 a.m. at the Franklin County Hospital, Greenfield.

HAMPSHIRE
- March 3.
- May 5.

MIDDLESEX EAST
- January 27.
- March 17.
- May 5.

All meetings are held at the Bear Hill Golf Club, Stoneham, at 12:15 p.m., except that of May 5, which will be held at 6:30 p.m.

MIDDLESEX NORTH
- April 28.
- July 28.

PLYMOUTH
- February 18. Jordan Hospital, Plymouth.
- March 18. Goddard Hospital.
- April 15. Bridgewater State Farm.
- May 20. Lakeville.

WORCESTER
- February 10. Worcester State Hospital, Worcester.
- March 10. Memorial Hospital, Worcester.
- April 14. Hahnemann Hospital, Worcester.
- May 12. Annual meeting.

WORCESTER NORTH
- April 28.
- July 28.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Biological Symposia: A series of volumes devoted to current symposia in the field of biology. Edited by Jaques Cattell. Volume IX. *Sex hormones.* Edited by F. C. Koch, Ph.D.; Frank P. Hixon, professor and chairman of the Department of Biochemistry, University of Chicago; and Philip E. Smith, Ph.D., professor, College of Physicians and Surgeons, Columbia University. 8°, cloth, 146 pp., with 43 illustrations and 8 tables. Lancaster, Pa.: The Jaques Cattell Press, 1942. $2.50.

This monograph discusses the action and metabolism of the sex hormones and their relation to inversion of sex in the lower animals.

Sulfanilamide and Related Compounds in General Practice. By Wesley W. Spink, M.D., associate professor of medicine, University of Minnesota Medical School. Second edition. 8°, cloth, 374 pp., with 1 illustration, 18 charts and 6 tables. Chicago: The Year Book Publishers, Incorporated, 1942. $3.00.

This second edition has been thoroughly revised in the light of the rapid developments in the field of sulfanilamide therapy. The author has drawn on his experience with 2000 patients at the University of Minnesota hospitals.

(Books Received continued on page xv)

GRUNE & STRATTON
Medical Publishers
443 FOURTH AVE.
NEW YORK N. Y.

BARZILAI
Atlas of Ovarian Tumors
264 pp., 258 illus. $10.00

GOLDSTEIN
Aftereffects of Brain Injuries in War $4.00

LICHTWITZ
Functional Pathology $8.75

NEAL
Encephalitis $6.75

MILLER
Central Autonomic Regulations $5.50

FISCHER & WOLFSON

READY IN FEBRUARY

THE INNER EAR—INCLUDING OTONEUROLOGY, OTOSURGERY, and Problems in Modern Warfare

By Joseph Fischer, M.D., formerly, associate of Adam Politzer and senior otolaryngologist with Gustav Alexander, Policlinic of Vienna; staff member, Beth Israel Hospital, Boston, and **Louis E. Wolfson, M.D.,** instructor in ear, nose, and throat, Tufts College Medical School; senior surgeon in ear, nose and throat, Boston Dispensary.

This new book, based on twenty-five years of research, clinical experience, and postgraduate teaching, presents completely the various problems concerning the normal inner ear, its diseases, and their intracranial complications.

Written by two broadly trained otolaryngologists, the book covers thoroughly the anatomy, physiology, pathology, clinical tests, and treatment of the inner ear. Original illustrations and photomicrographs of postmortem material collected over a period of more than two decades accompany the text.

Special chapters dealing with war trauma, the role of the inner ear in aeronautics, and the inner ear in changes of atmospheric pressure have been made possible by Dr. Wolfson's experience in England during the blitz bombing of 1940-41.

510 pages **index** **bibliography** **77 illustrations, 4 in colors** **cloth, $5.75**

Hemolytic Syndromes. By William Dameshek, M.D., Tibor J. Greenwalt, M.D., Russell J. Tat, M.D., and Camille Dreyfus, M.D. A reprint of an exhibit sponsored by the New England Medical Center, Boston. Presented at the 1942 meeting of the American Medical Association, Atlantic City, June, 1942. F°, paper, 43 pp. Boston: The Bailey Press, $1.50.

A series of graphic charts depicting the various hemolytic processes.

First Aid: Surgical and medical. By Warren H. Cole, M.D., professor and head of the Department of Surgery, University of Illinois College of Medicine, and director of the Surgical Service, Illinois Research and Educational Hospitals, Chicago; and Charles B. Puestow, M.D., Ph.D., associate professor of surgery, University of Illinois College of Medicine and Graduate School, surgeon, Illinois Research and Educational Hospitals, senior surgeon, Henrotin Hospital, and consulting surgeon, Municipal Contagious Disease Hospital and Bethany Home and Hospital, Chicago, and St. James Hospital, Chicago Heights. 8°, cloth, 351 pp., with 92 illustrations. New York: D. Appleton–Century Company, Inc., 1942. $3.00.

A new book prepared especially as a teaching text for medical schools. It may also be used by physicians and nurses taking "refresher" or advanced first-aid courses. The subjects of wounds, burns, hemorrhage and shock are treated in considerable detail. The authors have particularly stressed treatment adopted by the American armed forces in the present war.

Manual of Dermatology. Issued under the auspices of the Committee on Medicine, Division of Medical Sciences, National Research Council. By Donald M. Pillsbury, M.D.; Marion B. Sulzberger, M.D.; and Clarence S. Livingood, M.D. 8°, cloth, 421 pp., with 109 illustrations and 11 tables. Philadelphia and London: W. B. Saunders Company, 1942. $2.00.

A manual containing the methods officially approved for use by the Medical Department of the United States Army. It is confined to the diagnosis and treatment of skin diseases.

When Doctors Are Rationed. By Dwight Anderson, director of public relations, Medical Society of the State of New York; and Margaret Baylous, therapist, Charleston General Hospital, Charleston, West Virginia. 12°, cloth, 255 pp. New York: Coward–McCann, Incorporated, 1942. $2.00.

A popular book for the laity, describing the duties of the Procurement and Assignment Service in Washington, with suggestions to patients on their attitude toward the physician during the present emergency.

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(*Hospital Literature*)

The swaddled infant pictured at right is one of the famous works in terra cotta exquisitely modeled by the fifteenth century Italian sculptor, Andrea della Robbia. In that day infants were bandaged from birth to preserve the symmetry of their bodies, but still the gibbous spine and distorted limbs of severe rickets often made their appearance.

A bambino from the Foundling Hospital, Florence, Italy,—A. della Robbia

Glisson, writing in 1671, described an ingenious use of swaddling bands — "first crossing the Brest and coming under the Armpits, then about the Head and under the Chin and then receiving the hands by two handles, so that it is a pleasure to see the Child hanging pendulous in the Air . . . This kind of Excercise . . . helpeth to restore the crooked Bones. . . ."

STRAPPED FOR RICKETS

SWADDLING was practised down through the centuries, from Biblical times to Glisson's day, in the vain hope that it would prevent the deformities of rickets. Even in sunny Italy swaddling was a prevailing custom, recommended by that early pediatrician, Soranus of Ephesus, who discoursed on "Why the Majority of Roman Children are Distorted."

"This is observed to happen more in the neighborhood of Rome than in other places," he wrote. "If no one oversees the infant's movements, his limbs do in the generality of cases become twisted. . . . Hence, when he first begins to sit he must be propped by swathings of bandages. . . ." Hundreds of years later swaddling was still prevalent in Italy, as attested by the sculptures of the della Robbias and their contemporaries. For infants who were strong Glisson suggested placing "Leaden Shooes" on their feet and suspending them with swaddling bands in mid-air.

How amazed the ancients would have been to know that bones can be helped to grow straight simply by internal administration of a few drops of Oleum Percomorphum. What to them would have been a miracle has become a commonplace of science. Because it can be administered in drop dosage, Oleum percomorphum is especially suitable for young and premature infants, who are most susceptible to rickets. Derived from natural sources, this product is rich in vitamins A and D. Important also to your patients, Oleum Percomorphum is an economical antiricketic.

EXIGENCY OF WAR

Oleum Percomorphum 50% is now known as Oleum Percomorphum 50% With Viosterol. The potency remains the same; namely, 60,000 vitamin A units and 8,500 vitamin D units per gram. It consists of the liver oils of percomorph fishes, viosterol, and fish liver oils, a source of vitamins A and D in which not less than 50% of the vitamin content is derived from the liver oils of percomorph fishes (principally Xiphias gladius, Pneumatophorus diego, Thunnus thynnus, Stereolepis gigas, and closely allied species).

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